VI.—*The Spread of the Excitatory Process in the Vertebrate Heart.* Parts I–V.

By Thomas Lewis.*

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Part I.—*The Toad's Ventricle.*

[Plates 15 and 16.]

Introductory.

If non-polarisable electrodes are placed upon the body of the toad, the one contact lying on the muscle near the root of the neck in front and on the right side, while the other contact rests on the muscle in the centre of the abdominal wall, and these contacts are united to the string galvanometer in such a way that relative negativity of the cephalic contact yields in our photograph an upward excursion; then the form of the curve which is written at each cycle of the heart may be exemplified by the upper curve of fig. 1 (next page).

This curve is representative, and is a record of events which are almost uniform when the heart is beating normally and its rate is about 25 beats to the minute. It consists of an upward, rounded, and sometimes subdivided deflection, P; this deflection is followed by a period of rest, during which the string lies on the zero line or near it. Ventricular activity is first signalled by the summit R, and this elevation invariably begins in a gradual fashion, its steepness increasing as it proceeds. The string, after a variable interval of from 0·0400 to 0·0900 second, falls back towards the base line. It may or may not attain that line, but it usually does, and, travelling somewhat beyond, produces a diminutive deflection S; this ends the first part of the ventricular complex. Then follows a second period of rest, which is finally disturbed by a slow and upward movement of the string in the inscription of the deflection T. In many cases a notch, B, is found in the interval between S and T.† (The little deflection B has usually a downward direction, and is the result of activity in the *truncus arteriosus*.)

Such is the customary and almost constant form of the axial electrocardiogram in an extracardial lead, and, by common consent, P represents activity of the auricle,

* Working under the Medical Research Committee. A preliminary report of the observations appeared in the 'Proceedings of the Physiological Society,' June 5, 1915.

† The P–R interval and the R–B interval in this curve are of more than average duration.

while the remaining deflections represent the activities of the ventricle. Of these deflections, R and S* may be regarded as expressing the spread of the excitation wave in the ventricle; the horizontal line between S and T' may be regarded as expressing equal activity of all parts of the ventricle, and T' may be regarded as expressing the decline of the excitatory stage. But until recently these axial extracardial curves have received little attention. In earlier works the contacts were placed upon the muscle of the heart. The curves which SANDERSON and PAGE† took with the capillary electrometer were from contacts placed upon the base and apex of the ventricle. These writers state‡ that when such a lead is adopted two deflections are recorded, the first of which indicates basal negativity (an upstroke), and the second of which indicates apical negativity (a slow downstroke). The two deflections of opposite sign described by SANDERSON and PAGE have been recorded by many experimenters, and out of this observation the impression has grown that the excitation wave proceeds as a simple muscle wave from base to apex of the heart. But recently it has been ascertained by SAMOJLOFF§ and GOTCH‖ that in certain base-apex leads the second slow deflection has the same direction as the primary deflection, and GOTCH, who appears to have been prompted in the first place by this fact, ventured the hypothesis that the excitation wave travels from base to apex and ultimately returns to the base. He found confirmation of this view in an inconspicuous movement of the recorder in a downward direction, at a time corresponding somewhat closely to S in fig. 1. Briefly, the base-apex curve was said to be triphasic, comprising (1) a base-negative effect, (2) an apex-negative effect, and (3) a base-negative effect.

It is requisite, before proceeding further, to examine this direct method of leading more closely. In fig. 1 are two curves, the upper of which has been described.

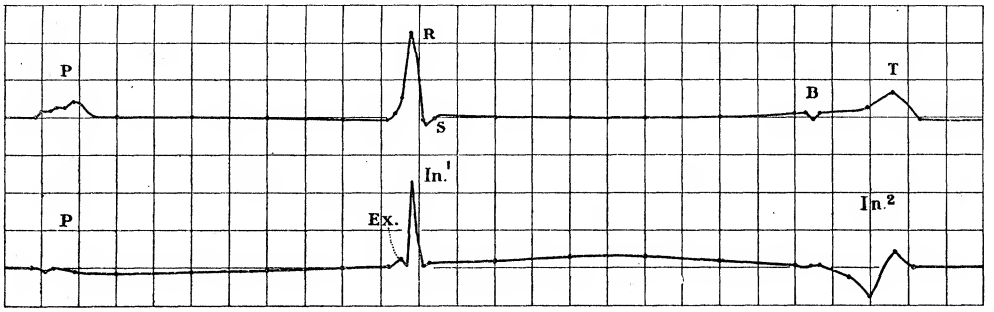


FIG. 1.—A chart constructed from two simultaneously written curves—the upper from an extracardial base-apex lead, the lower from a direct base-apex lead. The abscissæ represent tenths of seconds; the ordinates of the upper curve are on the scale of 2 millivolts to the centimetre (two scale divisions), and of the lower curve 2 millivolts to the *millimetre*.

* The amphibian electrocardiogram shows no Q.

† 'Journ. of Physiol.,' vol. 4, p. 327 (1883–84).

‡ 'Roy. Soc. Proc.,' vol. 27, p. 410 (1878).

§ 'Archiv f. d. ges. Physiol.,' vol. 155, p. 471 (1914).

‖ 'Roy. Soc. Proc.,' vol. 79, p. 323 (1907).

Below it is a simultaneously written curve, inscribed from a base-apex lead such as SANDERSON and PAGE and GOTCH employed. Upon cursory inspection it presents similar features to the upper curve, and the deflections In^1 and In^2 are the deflections recorded by these workers and recognised by physiologists ever since their papers were published.

But when we analyse the two curves more carefully we find they differ in certain very essential particulars. In the first place, the sensitivity of the recording fibre of the upper curve was ten times as great as that of the lower curve, and this greater sensitivity in the case of the extracardial lead was not necessitated by the greater resistance in circuit, for by the method of standardisation this extra resistance is taken into account.*

We may say, therefore, that the potential difference developed in the lower curve at the time R was written was ten times as great as in the upper curve.

But the curves differ in another very essential respect. The upstrokes R and In^1 are not simultaneous; R has a start of some 0·0450 second.† The first two-thirds of the upstroke R are written before In^1 starts, and the latter has a far steeper rise than the former. Consequently, we may affirm that these upstrokes are not due to the same events. Moreover, the deflection In^1 is preceded by a summit Ex, which has no counterpart in the upper curve.‡

These and other observable differences are due to the change of lead, and may be explained in the following manner. In the extracardial lead the whole heart lies between the electrodes; the relation of the upper contact is very similar to all parts of the heart's base, and the relation of the lower contact is very similar to each part of the apex. The curve represents as fully as any single curve can the sum total of the heart's activities and the individual activities proportionately. But where the contacts are placed on the muscle at base and apex, the points chosen for contact are favoured points, and our curve then consists of (1) a general expression of the activities, and (2) a special expression of the activities of muscle beneath the contacts. In a paper written with MEAKINS and WHITE§ I have shown that the curves of direct leads comprise effects of these two kinds, which are termed *extrinsic* and *intrinsic*, respectively. The intrinsic effects are those signalling events which occur in the immediate neighbourhood of the contacts; the extrinsic effects at some distance from them. In all direct leads from the heart, whatsoever the method employed, these two sets of effects are intermixed and are often very difficult to disentangle. Until complete separation is possible, and until the extrinsic effects are

* With circuit resistances of from 20,000 to 40,000 ohms the instrument may be regarded as a potentiometer; standardisation is obtained by throwing a known E.M.F. into circuit and obtaining for that E.M.F. a given excursion.

† Measurements made upon the original negative by comparator, as in previous papers.

‡ In curves taken with the capillary electrometer these small initial phases seem to have been lost.

§ 'Phil. Trans.,' B, vol. 205, p. 375 (1915).

more fully understood than they are at present, we cannot be too circumspect in interpreting curves taken by leads direct from the heart muscle.

Adapting our terminology to fig. 1, we may describe the whole upper curve as uniformly extrinsic in origin. In respect of the lower curve, *P* the auricular deflection is evidently extrinsic. The initial ventricular deflection *Ex* is also extrinsic. I have been at pains to show, by observations upon the dog's auricle and ventricle, that the first intrinsic deflection signals the arrival of the excitation wave immediately beneath one contact, and it may be recognised by its direction and by its steepness. Thus, when the wave of activity is prevented from reaching the contact, this deflection no longer appears in the curve; the later deflections, represented in our present curve by In^2, are abolished at the same time. These deflections are due to some local change, while *P* and *Ex*, unaffected by the same procedure, are due to events occurring at a distance.

How much of the initial movement In^1 is due to local change under the basal contact, and how much to local change under the apical contact, and, further, to what extent it is complicated by hidden extrinsic effects, become matters for enquiry. These questions emphasise the precise difficulties which confront the interpretation of each direct lead from two contacts placed upon the ventricle. Move the extra-cardial contacts to one or other side over a considerable range and there is little perceptible change in the curve; move one of the direct contacts ever so little and change of form, often profound, is the almost invariable result. The lower curve of our figure is not characteristic of a direct base-apex lead in the toad; no such curve is characteristic; curves of many shapes, and separate curves with the chief deflections in opposite directions, may be obtained in one and the same animal. So changeable is the form, and so varied and so many-phased, that deductions from such leads are often precarious and may be most misleading. The present communication deals only with the preliminary phases of the ventricular curve, represented in fig. 1 by *R* and *S*, to which attention may now be confined. The animal which gave the base-apex curve of fig. 1 also gave, by a slight movement of the basal contact, the base-apex lead of fig. 2, *I*. Here the initial phases are alone plotted, and on a different scale, but the wide departure from the initial phases of fig. 1 (*Ex*, In^1) is at once manifest. Some insight into the composite character of this curve may be gained by studying the curves from the companion leads, illustrated below the chart. The three curves of the chart are plotted on a uniform scale and with the corresponding time phases vertically above each other. The three contacts used were (1) base, (2) apex, (3) body wall, and the leads were arranged around the sides of a triangle (*I*, base to apex; *II*, base to body wall; and *III*, apex to body wall). Now Leads *II* and *III* represent our accustomed method of leading direct from the heart; one contact lying on the muscle, the other on an indifferent point; and such curves resemble each other in one respect; after the inscription of certain initial phases, usually downward in direction and relatively slow in movement, they are broken by

a steep rise, which signals relative negativity of the corresponding heart contacts. As we find that this rise corresponds to the arrival of the excitatory process beneath the contact, the advantage of placing a single contact upon the ventricle is clear; we avoid the clashing of local events under two contacts of a pair when each contact lies on the muscle. By our method, the direction which the deflection, signalling the arrival of the excitation process, will take is known beforehand; such deflections are steep upstrokes. Leading thus from base and apex, each to body wall, the corresponding intrinsic deflections are sharply defined, and we observe in the present instance that the apex becomes active before the base (actually by 0·0070 second). We may now read into the *base-apex* lead the same event, the first sharp *downstroke* of this curve is evidently due to relative negativity of the apical, as opposed to the basal, contact, for it occurs at almost the same instant as the upstroke of the apex to body wall lead. But if we read the first curve upon the accustomed plan we must acknowledge that the first event in the base-apex lead is an indication of relative negativity of the base, for a rise precedes the fall. The initial phases of this curve are clearly of the same origin as the initial downstrokes of Leads *II* and *III;* they are extrinsic effects. *Thus, we are not justified in concluding from the direction of the first deflection in the base-apex lead, that base or apex first becomes active.* Neither are we justified in using the first prominent deflection of such a lead as an indication, for it not infrequently happens that the chief extrinsic deflection is as prominent as the intrinsic deflection. We *are* justified in using the most prominent upward deflection of leads such as *II* and *III* as indications, providing certain precautions are observed in doubtful instances. These remarks lead up to my final observations. My view is distinctly that the base-apex lead or any lead comprising two contacts on the ventricular surface must for the moment be abandoned in favour of other methods.

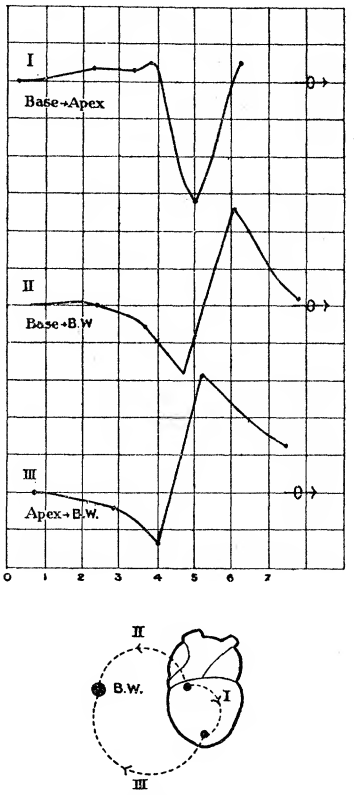


FIG. 2.—A chart of three curves taken from the same animal as fig. 1, but representing initial phases of ventricular curves only. The abscissæ represent hundredths of a second; the ordinates 1 millivolt to the millimetre. The curves are acceptable because completing a triangle they obey the formula $I + III = II$ (see p. 234). Below is an outline of the heart and corresponding leads to scale. *B.W.* = Body wall.

CLEMENT,* who hitherto has alone attempted to map out the course of the excitation wave in the frog's ventricle, has adopted a special method of leading. He uses a thread bent at an angle, the ends of which are in circuit with the recording

* 'Zeitschrift f. Biol.,' vol. 58, p. 110 (1912).

instrument; he applies the angle of the bent thread to the surface of the heart, and thus obtains an ingenious form of electrode of which the two contacts may be regarded as in very close approximation. Nevertheless he does not avoid the pitfalls of such direct double contact leads; his curves clearly show the presence of extrinsic effects*; many are diphasic, consisting of a preliminary extrinsic and a final intrinsic deflection of almost equal extent; to my mind his deductions, both from the direction of the prominent deflection and from the time at which it has its beginning, are open to very serious objections; and the discrepancies between his conclusions and those which I now propose to narrate are to be explained by the difference in our method of observation.

Surface Distribution of the Excitation Process.

The observations upon which this report is based were made on large female toads of the species *Bufo vulgaris major* in the months of March and April. The heart rates in different experiments varied from 20 to 29 per minute. The toads were pithed, brain and cord being completely destroyed; the vagi were left intact.

Readings of the commencement of the excitatory process were taken by the method already described by LEWIS and ROTHSCHILD,† the sole difference in procedure being a reduction in the size of electrodes. For direct contacts I have used small bore glass tubing filled with kaolin, and an upper layer of copper sulphate; a cotton thread daubed with moist kaolin and freshened from time to time formed the actual contact; this was paired with a contact on the body wall.

The hearts have been drawn to scale *in situ*, and the outlines of the figures are reproduced at natural size.

In all illustrated experiments the curve of Lead *II* has been used as a standard of measurement. I do not use the upstroke of *R* for this purpose, but its summit, for the commencement of the curve is slow and cannot be estimated with sufficient constancy. The readings as given in this paper are all related to the commencement of *R*, however; the interval between upstroke and summit being measured in a number of plates, and the average interval being allowed in the calculation of final readings (Plate 15, fig. 7).

The Ventral Surface.—Readings from the ventral surface of the heart are illustrated by figs. 3, 4, and 5. In a given animal the total variation in the intervals is from 0·0150 to 0·0350 second. The excitation wave does not appear simultaneously over the ventral surface, as CLEMENT has stated. The order of the readings is variable: almost every rule which may be formulated is broken by frequent exceptions.

* I would point out that CLEMENT'S control of his method is an experiment upon the sartorius muscle, where conduction is upon a simple plan and where the confusion of the extrinsic deflection is not encountered.

† 'Phil. Trans.,' B, vol. 206, p. 181 (1915).

In three instances all basal readings, *i.e.*, those taken along the *A–V* groove, are higher than the extreme apical reading (figs. 3 and 5, *E* and *F*), by values of 0·0100–0·0300 second or more; in two other instances one or more of the basal readings is somewhat higher than the extreme apical reading (fig. 5, *D* and *G*). In three instances (fig. 4) all or most of the basal readings are lower than the apical reading.

The belief that the base of the heart is activated before the apex is therefore

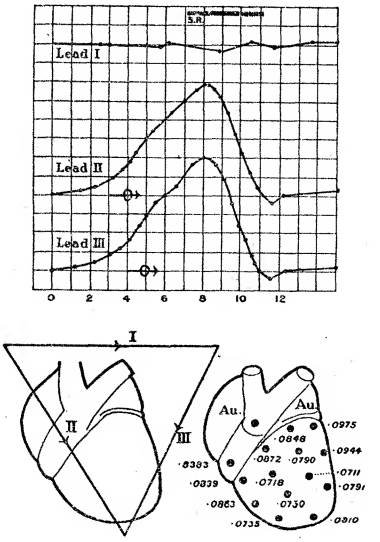


FIG. 3.

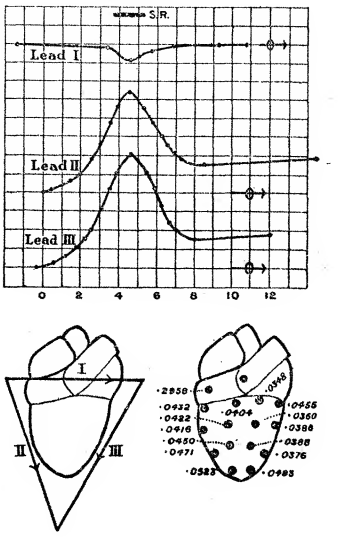


FIG. 4.

FIG. 3.—A chart showing the initial phases of the electrocardiogram in extracardial leads *I*, *II*, and *III* of Toad *B*. Ordinates, 1 cm. = 1 millivolt; abscissæ, 1 cm. = 0·0400 second. Also two natural size outlines of the corresponding heart to scale. The surface readings of the right-hand figure are accurately related to the commencement of *R* as it is charted. The readings of the deflections in the chart and the surface readings of the diagram may be compared therefore. The left-hand figure shows the relation of the three lead contacts to the heart. The charts of this and the succeeding figure have been re-drawn and are not precisely accurate.

FIG. 4.—A similar chart and outlines of the heart of Toad *C*. The scale of ordinates in this figure is such that 3 cm. = 1 millivolt; abscissæ as in previous figures. The surface readings of the right-hand figure are accurately related to the commencement of *R* as it is charted. The readings of the deflections in the chart and the surface readings of the diagram may be compared therefore.

without foundation for the majority of toads; the reverse is usually the case. Neither can GOTCH'S conclusion that the wave starts from the base, travelling to the apex and returning to the base, be entertained. It is the rule that some point in the central region always has precedence to base and apex. The earliest region of the ventral surface to become active is the central zone, sometimes actually the centre point (figs. 3 and 5, *E* and *F*) or a point very close to it; but in three instances the earliest point has been towards the left or right margin of the central zone. In only one instance has the earliest point lain outside the central

zone (fig. 4), and in this instance it preceded the earliest point in the central zone by 0·0012 second only.

The left margin of the heart is usually activated either a little before the right margin, or the readings on the two margins are similar.

The Lateral and Posterior Surfaces.—In four animals (fig. 5, *D*, *E*, *F*, *G*), after having taken a series of readings from the ventral surface, the heart has been rotated so as to lie on its side and a series of additional readings has been taken, overlapping the fields of observation. The readings are of much the same order in magnitude as over the ventral surface and show similar variations. Here again the lowest reading is often discovered in the central zone (exceptions will be found in Toads *E* and *G*). In all instances (*D*, *E*, *F* and *G*) the base is as late or later than the apex.

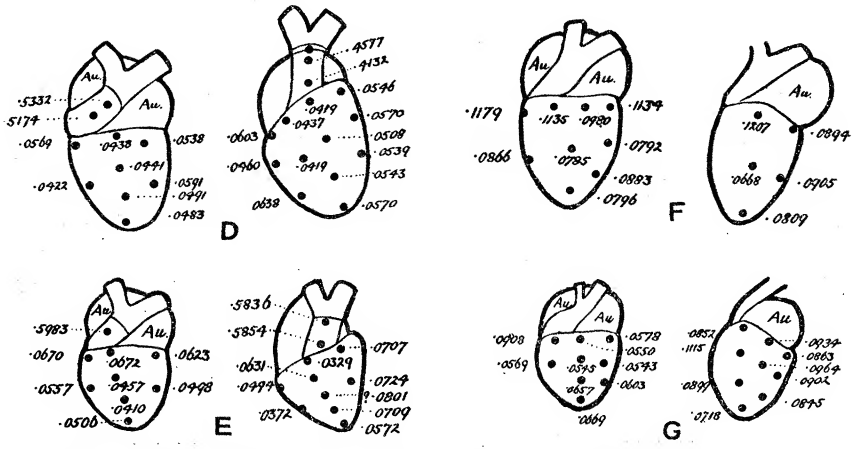


FIG. 5.—Natural size. Outline diagrams to scale of the hearts of Toads *D*, *E*, *F*, and *G*, showing a number of surface readings, expressed relative to the commencement of *R*. In this figure, also, the readings are related to the commencement of *R*; the intervals between the commencements and summits of *R* (the actual standard in measuring) will be found in Table I.

The left-hand outlines of the four paired figures are of the ventral surface of the heart in each instance; of the right-hand outlines those of *D* and *E* are of the right surface, those of *F* and *G* are of the left surface of the heart.

A number of readings from the *truncus arteriosus* are given in the illustrations. This tube is muscular in its first portion, and the limits of the musculature are readily to be recognised in the exposed heart. Contacts placed on the muscle give very late readings. As a general rule, the excitation wave reaches the *truncus arteriosus* a half second after the whole surface of the ventricle is involved. The interval is variable, extending from 0·25 to 1·0 second. The surface change to the high reading is abrupt at the line of junction with ventricle, and there appears to be a natural blocking point between ventricle and truncus. That the excitation wave travels up the truncus may be shown by obtaining readings from the upper and lower ends of the muscular tube. If such readings are taken by separate comparison with the standard curve (as in the figures) the reading from the lower reaches of the conus is usually the lesser value; but it may be of equal value with the reading from the higher reaches of the truncus

(as in fig. 5, *E*). The method is insufficient as the ventricle-truncus interval is great, and subject to correspondingly large fluctuations (compare ventral and side readings in fig. 5, *D*). The order of excitation is conclusively shown to be in the direction stated by simultaneous curves taken from two truncus contacts ; in these observations the progression is invariably in the cephalic direction (see Table III).

The *truncus* activity is often signalled in the axial electrocardiogram (see fig. 1, and Plate 15, fig. 8), being represented by a shallow dip which falls between *R* and *T*. A lead from the truncus yields two intrinsic phases (Plate 15, fig. 8), *B* the intrinsic deflection proper (see Plate 16, fig. 10), and *BT* the final effect, which is of a similar nature to *T* in the axial curve. It also yields large extrinsic effects from auricle and ventricle (fig. 8, *R* and *T* of lower curve). If the contact is moved off the muscle and on to the artery, the remaining conditions being constant, the intrinsic effects are lost, while extrinsic effects remain (Plate 16, fig. 9).

The Relation of Surface Readings to the Axial Electrocardiogram in Extracardial Leads.

This may be studied in figs. 3 and 4 and by a comparison of the remaining figures with Table I. In figs. 3 and 4, which may be taken as fair illustrations, the initial phases of the electrocardiogram in Leads *I*, *II* and *III* are charted relative to the commencement of *R*. Below the chart the 1/100 second time lines are enumerated ; the commencement of *R* stands on the 0 line. All the surface readings fall within the space bounded by the thick line *S R* on the charts. The ventral surface begins to be activated during the time phase which corresponds to the last third of the upstroke *R*, and other points are activated while the summit and first half of the downstroke is written. As the initial phases of the electrocardiogram (*R* and *S*) are supposed to correspond to the activation of the muscle, we enquire why the surface readings do not cover the same time period as the complete initial deflections of the axial curve.

TABLE I.

Toad.	Average heart rate.	R.*	Summit of *R*.	Descent of *R*.	Bottom of *S*.	Return of *S*.	Latest intrinsic reading.
A	37	0·00	0·0836	0·1030	0·1100	None	—
B	24	0·00	0·0828	0·1100	0·1158	0·1220	0·0975
C	26	0·00	0·0454	0·0800	None	—	0·0523
D	29	0·00	0·0618	0·0889	0·0917	0·0990	0·0638
E	27	0·00	0·0429	0·0531	0·0668	0·0736	0·0724
F	27	0·00	0·0725	0·0913	0·1005	0·1243	0·1207
G	26	0·00	0·0688	0·0914	0·1018	0·1408	0·0964
H	25	0·00	0·0885	0·1271	None	—	0·0776
J	24	0·00	0·0682	0·0997	None	—	0·0829
K	20	0·00	0·0505	0·0676	None	—	0·0495

* All readings of initial phases apply to Lead *II* only, and are related to *R*.

The hiatus which coincides with the first two-thirds of *R*'s upstroke we may neglect for the moment. The shorter hiatus, corresponding to the end of the downstroke of *R* and to *S* is readily explained. The readings on the dorsal surface of the heart are in general a little higher in value; this difference may be in small measure responsible. It may be also that the surface of the ventricle which curves into and lies buried in the *A–V* groove is activated later; that such is the case would be anticipated from the usual order of readings; in brief, the latest surface readings are probably not obtained, as the whole muscle is inaccessible. But a certain and chief cause of the hiatus is that our readings are taken from the beginnings of intrinsic deflections, or of a time instant when the surface potential is beginning to develop. How long it continues to develop is uncertain, but, judging from the time taken for intrinsic deflections to reach their full amplitude, time intervals of 0·0100–0·0150 second must be allowed. If we add such time intervals to the readings of the latest intrinsic deflections in the last column of Table I, we shall find close correspondence between the figures at which we arrive and the end of the initial deflections in the axial lead, and so may draw the general conclusion that the *surface excitation is coincident with the initial phases of the axial electrocardiogram in extracardial leads from a point well up the upstroke of R to the termination of these phases.*

Further Observations.

When we consider the surface readings we may conclude from these alone that the excitatory process does not travel as a simple surface wave. For, if that were so, not only would the readings be arranged in orderly succession, but the simultaneous appearance of the wave at points far removed from each other would not be discovered, unless, indeed, spread occurred from a central surface point. But such readings as we possess are not compatible with any such simple mode of surface spread.

To test this question more fully I have made a number of observations upon surface conduction rate.

I have used two direct contacts, *p* and *d*, each paired with an indifferent contact on the chest wall, just as ROTHSCHILD and I proceeded in investigating the dog's heart. Stimulation* is in line with *p* and *d*, the heart contacts of the proximal and distal leads. The interval between the two intrinsic deflections written in the simultaneous curves is estimated and the distance between *p* and *d* is measured. My results are given in Table II, the arrows in the last column indicating the direction relative to the ventricle as a whole in which the excitation wave travelled past the contacts in different experiments. With a solitary exception the calculated rates of transmission are sufficiently uniform, yielding an average of 76 mm. per second.

We may compare this rate of conduction of artificial waves propagated over the ventricle with the natural rate of conduction over the *truncus arteriosus* (Table III).

* In the estimation of rate, two or more of the first four beats of a succession of responses were alone utilised.

TABLE II.—Rates of Conduction.

Toad.	Interval.	Distance.	Rate.	Average rate.	Direction of conduction.
	sec.	mm.	mm. per sec.	mm. per sec.	
F.	0·0984*	4	41	42	↖
F.	0·1171	5	43		↗
G.	0·0491	3·2	65	89	↑
G.	0·0252	2·8	111		↖
G.	0·0558	5	90		↗
H.	0·0704	5	71	65	↗
H.	0·0681	4	59		↖
H.	0·0187	6	311†	—	→
J.	0·0507	4·2	83	88	↖
J.	0·0610	4·8	79		↗
J.	0·0417	4·2	101		←
K.	0·0408	5·2	127	96	↗
K.	0·0655	4·3	66		→

Average rate of conduction, 76 mm. per sec.

* Each figure is an average of two readings.

† Excluded from averages.

NOTE.—It is to be regretted that the temperature of these animals was not taken; the observations were undertaken during mild weather, and the laboratory temperature was usually about 16–18° C.

TABLE III.—Natural Conduction Rates in *Truncus arteriosus*.

Toad.	Interval.	Distance.	Rate.
	sec.	mm.	mm. per sec
D	0·0173*	2	116
D	0·0253	3	119
J	0·0152	2·8	184
K	0·0240	2·4	100

Average rate of conduction, 130 mm. per sec.

* Each figure is an average of two or more readings.

These estimated rates are higher, averaging 130 mm. per second. An over-estimating of rate is to be anticipated in the *truncus*, for, utilising the natural excitation wave,

its precise line of travel is unknown, and any deviation of the line of contacts from the line of travel will of necessity yield an error in this direction.

We may take 100 mm. per second as a safe approximation of the rate of travel in ventricular muscle.*

On this basis, the basis of calculated rate of conduction in the animals concerned, a simple base-apex or apex-base spread in a ventricle having a length of 13 mm. (fig. 3) would have a duration of 0·13 second, over a ventricle of 17 mm. (Toad *A*) of 0·17 second. The complete spread actually occupied 0·0233 and 0·0369 second respectively in these animals. The actual rate of spread is five times greater than can be accounted for in assuming spread from base or apex, and two and a half times greater than can be accounted for in assuming spread from a central focus.

The relative rapidity of spread and the general order of surface readings led me to anticipate a similar form of distribution to that discovered in the dog's heart, namely, spread from within outwards. And the conclusion that such is indeed the mode of distribution was strengthened both by the architecture of the toad's ventricle and by the failure of surface readings to account for the first deflection *R* of the axial electrocardiogram.

Internal Readings.

To obtain readings from the lining of the ventricle I have used a glass tube drawn out at the end to a bore of $\frac{1}{2}$ mm., and suitably curved for introduction through a slit in the wall of the left auricle. The end of this tube is plugged with the kaolin, the body of the tube is filled with copper sulphate.

Nine readings have been taken from five toads, the electrode being introduced well into the cavity, and these are given in Table IV, in which the earliest external reading is also given in an extra column. These readings, ranging from 0·0047 to 0·0572 second, are variable, as is to be expected in a musculature of this extent and complexity. With a solitary and unimportant exception, they are all earlier than the earliest external reading in the corresponding animals.

In three of the toads the contact was placed just inside the ventricle near the *A–V* ring. These additional readings are the very earliest obtained, 0·0005, 0·0012, and −0·0094, and approximated closely to the beginning of *R*. It should be stated that the intrinsic deflection in these three curves is relatively blunt at its origin, and the reading is therefore subject to greater error.†

The bluntness is ascribed to blending of extrinsic and intrinsic deflections, a phenomenon which was witnessed by Meakins, White, and myself, when, in studying the dog's auricle, we led off from the region of the pacemaker.

I conclude, therefore, that the excitation wave spreads from the ring and passes outward to the surface through the wall of the heart.

* Sanderson and Page estimated the rate at 125 mm. per second. Clement estimates it at 320 mm. per second, but this value is clearly too high.

† But probably no more, however, than 0·0030 second.

TABLE IV.

Toad.	Figure.	Lowest external reading.	Number of external readings.	Internal readings.	Internal readings (near ring).
F	5	0·0668*	14	0·0377 0·0572	—
G	5	0·0543	18	0·0281	—
H		0·0391	4†	0·0047 0·0282	0·0005‡
J		0·0579	4†	0·0092 0·0132	0·0012§
K		0·0227	8†	0·0073 0·0254	0·0094§

* All readings are averages.

† Points specially chosen as probably including the earliest reading, *i.e.*, the central zone.

‡ Inside ring in front at left auriculo-ventricular border.

§ Inside ring in front near conus exit.

The Excitation Wave in the Ventricle as a Whole, and the Interpretation of the Curves of Extracardial Leads.

That the spread of the excitation wave in the toad's heart is in general from base to apex is a conclusion which is suggested by the form of electrocardiogram yielded by axial extracardial leads. The direction of the first and chief deflection *R* (fig. 1, and Plate 16, fig. 11) is such as to indicate primary base-negativity. This conclusion has also been drawn from observations upon the surface of the heart, though fallaciously, for, as we have seen, the surface basal muscle is, as a rule, the last to be activated. We have, then, this curious paradox; evidence of a general base-apex movement of the excitatory process, and yet a movement on the surface of the heart, over its cephalic half at all events, in a contrary direction. At this stage I would refer once more to the first hiatus between the surface readings and the initial phases of the axial electrocardiogram. *The surface excitation has not begun until the greater part of R is written; this phase of the curve, taken from an extracardial lead, results from the excitation of the deep musculature.*

In attempting further to elucidate the extracardial curves, it has seemed to me essential that they should be studied on a broader basis than has hitherto been attempted. A single lead from contacts placed away from the heart provides but an uncertain index of the direction of the excitation wave. It is necessary in the first instance, by adopting several leads, accurately to determine the electrical axis of the heart in a given plane.

Method of Estimating the Electrical Axis.—The method adopted to estimate the

electrical axis in the present work has been that first described by EINTHOVEN, FAHR and DE WAART.* In using three leads, *I*, *II* and *III*, the contacts of which are arranged in the form of an equilateral triangle, the electrical potential developed by the heart is distributed on these lines according to the inclination of the electrical axis to them; and if the potential values expressed in the several leads are known, the direction of the electrical axis may be calculated trigonometrically.

Experimental Procedure.—Three contacts are arranged around the heart, being attached by stitches to the parietes of the unbroken body wall. The contacts are so placed as to form, so far as possible, the apices of an equilateral triangle. Two of the contacts lie on a line drawn horizontally across the body, usually on a level with the pectoral girdle, the third lies below the xiphisternum, near the heart's apex. Evidently the electrical axis will be calculated with the closest approach to exactitude when the triangle is exactly equilateral; but the error introduced by considerable deviations from the equilateral position is not very appreciable. Curves are taken from the three pairs of contacts, leading in the directions shown in the illustrations (figs. 3 and 4); and each of these curves, being of standard magnitude, is written simultaneously with a signal curve from a fourth lead (usually the floor of the mouth to a point on the right abdominal wall, see Plate 16, fig. 11). The three curves are measured on the comparator and plotted on millimetre paper, so that corresponding phases in time lie above each other vertically. A set of curves is accepted only if it obeys EINTHOVEN's formula throughout, that is to say, if, on any vertical line, the value of Lead *I* plus that of Lead *III* = the value of Lead *II*. The advantages of this procedure are manifold. The exclusion of curves which do not fit enforces accuracy of standardisation and of measurement; it eliminates all curves in which there are material errors in the estimation of time intervals or potential values. It checks in an admirable manner the factors of the apparatus employed; for a sufficiently quick response, uniformity of excursion with successive increments of potential within the working range, non-polarisability of the electrodes, are all essential to success. In brief, the fit of these charted curves assures us that each accurately portrays the current discharges which are tested, and that all are charted in correct time relations to each other. No material error in the calculation of angle is possible, except where, on a given vertical, all three curves nearly approach the base line of zero potential; at such points minute errors are of consequence and calculated angles may not be treated with the same confidence. Two such sets of plotted curves are given in figs. 3 and 4; and the relative values, expressed as potential, on successive 1/100 second time lines are given in the accompanying table† (Table V). The calculated angles formed by the electrical axis at the same time intervals are also given.

* 'Archiv f. d. ges. Physiol.,' vol. 150, p. 275 (1913).

† It will be noticed that small corrections have here and there been introduced into this Table; this has been done for the purpose of bringing them into perfect agreement with EINTHOVEN's formula.

TABLE V.

Toad B.

Seconds.	I.	II.	III.	Angle.
				°
0·0100	0	1	1	90
0·0200	0	2	2	90
0·0300	0	4	4	90
0·0400	0	8	8	90
0·0500	−0·5	15	15 15·5	92
0·0600	0	20	20	90
0·0700	−0·5	25	25·5	91
0·0800	−1	29	30	92
0·0900	−1·5	24	25 25·5	93
0·1000	0	11	11	90
0·1100	0	0	0	
0·1150	0	−2	−2	−90
0·1200	0	−1	−1	−90

Toad C.

Seconds.	I.	II.	III.	Angle.
				°
0·0100	−0·5	2	2·5	101
0·0200	−0·7	5	6 5·7	96
0·0300	−1	13	14	94
0·0400	−3	24	27	96
0·0500	−3	25	29 28	96
0·0600	−2	17	19	95
0·0700	−1	15	15·5 16	93
0·0800	0	8	8	90
0·0900	0	7·7	7·7	90

Explanation of Table V.

In the "seconds" column and the columns marked *I*, *II* and *III*, the chief data of the charts of figs. 3 and 4 are tabulated.

The charts were originally constructed at twice their present dimensions, and consequently the ordinate values as given in columns *I*, *II* and *III*, and taken as millimetres, are double the present measurements of the chart, and double the actual measurements of the curves (see fig. 11).

The figures which are ruled out are the actual estimations; the figures placed beside them are the corrected values used in determining the angles.

In the fourth column, under "angle," the angle formed by the electrical axis with the horizontal (Lead *I*) is given (moving in clockwise direction from the right of the horizontal line).

Such deviations from exactitude as were present originally are not great; they are due to errors in measurement; as these are of insufficient magnitude to influence my conclusions, I have considered the attempt to reduce these errors (by re-measurement) less profitable than the construction of similar and additional charts from other animals.

The Direction and Movement of the Axis.—The electrical axis of the toad's heart, studied from one instant to the next, is remarkable in one respect. It lies almost in the line of the axis of the body and moves but little to one or other side of it. In Toad *A* it departed no more than 9° from the body axis; in Toad *B* no more than 3°, and in Toad *C* no more than 11°. It is for this reason that transverse leads (Plate 16, fig. 11, Lead *I*) are unfavourable; and for the same reason Leads *II* and Leads *III* are almost identical (fig. 11). During the stage in which *R* is written the electrical axis is inclined at an almost uniform angle of 90° to the horizontal; I interpret this observation as meaning that *during the whole of this period the movement of the excitation wave has an average and almost uniform direction from base to apex.* In some animals this direction is maintained throughout the whole period of the initial phases, in other and more numerous instances (Toad *B*, Table V) the direction of the axis becomes suddenly reversed, forming an angle of −90° or thereabouts with the horizontal; this change corresponds to the writing of *S* in Leads *II* and *III*; and I interpret it as being due *to a sudden reversal of the average direction in which the excitation wave travels, being now from apex to base.* To what is the change due? It is due, in my mind, to the last stage in the journey of the excitation wave, to the upward movement *at the base of the heart.*

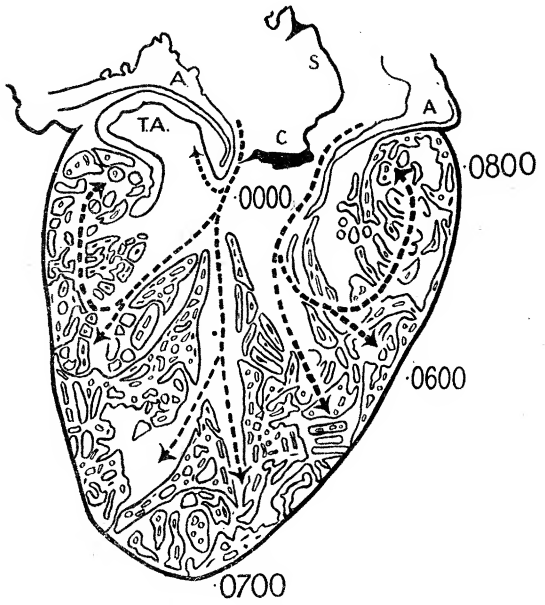


FIG. 6.—An outline drawing of a coronal section of a toad's heart, five times natural size. *A* = auricular muscle, passing into the *A–V* ring. *C* = endocardial cushion. *S* = auricular septum. *T.A.* = commencement of *truncus arteriosus.* The broken lines and arrows indicate the author's conception of the paths taken by the excitation wave. The readings (relative to *R*) in decimal points of seconds are intended to indicate the relative times at which the several muscular regions are activated.

My general hypothesis may be summed up diagrammatically. Fig. 6 represents a coronal section of the toad's ventricle, and illustrates the general arrangement of the musculature in the plane of our leads. The musculature of the auricle and ventricle does not meet at the surface *A–V* line, but the ring is prolonged as a tube for some distance into the ventricle; passing downwards it meets and fuses with processes of muscle which radiate towards the central surface zone and apex of the heart. While *R* is inscribed in the axial lead and the electrical axis is 90°, the excitation wave is spreading into the ventricle in many directions, but it is spreading more or less symmetrically and the average direction is down toward the apex. The slow onset of *R* is to be explained by the gradual and successive involvement of more and more

fibres continuous with the ring. The trabeculæ first carry the excitation to the central surface zone of the heart. Later, the wave courses farther toward the apex and at the same time mounts to the base. If the basal passage outlasts† the apical, the general direction of travel becomes reversed and the electrical axis, as expressed by the axial lead, becomes $-90°$. This hypothesis accords with the general distribution of internal and surface readings. The actual expression of the change to $-90°$ in an axial lead is the deflection *S*. I attribute *S* in axial leads in the toad to the final movement of the excitation wave over the base of the heart past the *A*–*V* groove. *S*, a deflection usually attributed to apical activity, appears to me in reality to be produced by a basal activity. That muscle activated at the base of the ventricle may be responsible for a dip in the curve, as opposed to a rise, has been shown already in the case of the *truncus arteriosus* (Plate 15, fig. 8, the dip marked *). It is not a question of the position of the muscle strip relative to the base or apex of the heart; it is a question of the direction in which it is activated, no matter where it lies. The axial electrocardiogram, as seen in an extracardial lead, must be regarded as a summation, an expression of the excitation of a large number of distinct muscle wedges, activated along separate routes. Each wedge brings, as a contribution to the total effect, its individual effect; the nature of its individual effect is controlled by two chief factors, the amount of muscle involved and the average direction in which the excitation wave passes through it.

Our conclusion in respect of *S* is justified by a comparison of the surface readings and Table I. In some axial curves from extracardial leads *S* is not developed; in the present series *S* was absent in Toads *C*, *H*, *J* and *K*. I have a sufficient number of surface readings in two of these animals; in Toads *C* and *K* the basal readings were of a lower order of magnitude than the apical readings. In Toad *A*, the basal and apical readings were similar, but descent below the base line was doubtful as the exact level of the latter could not accurately be determined. The remaining animals all presented *S* deflections of more or less prominence (*B*, *D*, *E*, *F* and *G*); these are the animals in which base readings came later than the extreme apical readings.

There seems, therefore, little reason to doubt that *S* in the axial lead from the toad's heart results from an upward passage of the excitation wave at the heart's base, and that the general distribution of the excitation wave is as I have depicted it in fig. 6.

There is, in the toad's heart, no evidence of a specialised conducting system in which the rate of conduction is peculiarly high. Evidence for this conclusion comes from three sources.

1. The rates of conduction, tested with artificial stimulation, are uniform over the whole ventricle, and are not dependent upon the architecture of the underlying structures.

2. The considerable time differences between separate readings from the lining of

† Or outweighs in virtue of the mass of muscle involved.

the ventricular cavity, in a given animal (Table IV), suggests a slow conduction rate over the lining.

3. The distance from the ring internally to the surface of the ventricular apex in the toads examined may be taken as a little less than 1 cm. The time taken for the excitation wave to travel over this path is approximately 0·0700—0·0800 second. The rate of travel along the lining is estimated therefore at about 100 mm. per second, a figure which does not differ materially from that calculated for the surface when the heart is excited.

Addendum.

TABLE VIII.—Extrinsic and Corresponding Intrinsic Deflections.

Toad A.		Toad B.		Toad C	
Extrinsic.	Intrinsic.	Extrinsic.	Intrinsic.	Extrinsic.	Intrinsic.
0·0209*	0·0667	0·0250	0·0872	−0·0012	0·0432
0·0235	0·0497	0·0410	0·0839	−0·0019	0·0404
0·0156	0·0676	0·0242	0·0848	−0·0043	0·0348
0·0181	0·0377	0·0400	0·0975	0·0275	0·0455
0·0059	0·0527	0·0347	0·0944	0·0094	0·0386
0·0163	0·0746	0·0201	0·0791	−0·0029	0·0376
0·0223	0·0526	0·0150	0·0810	−0·0092	0·0493
0·0077	0·0371	0·0161	0·0735	−0·0072	0·0523
0·0096	0·0538	0·0456	0·0863	0·0048	0·0471
—	—	0·0354	0·0718	−0·0015	0·0416
—	—	0·0219	0·0730	−0·0071	0·0422
—	—	0·0202	0·0711	−0·0016	0·0360
—	—	0·0313	0·0790	−0·0045	0·0388

* Each figure is an average of two readings.

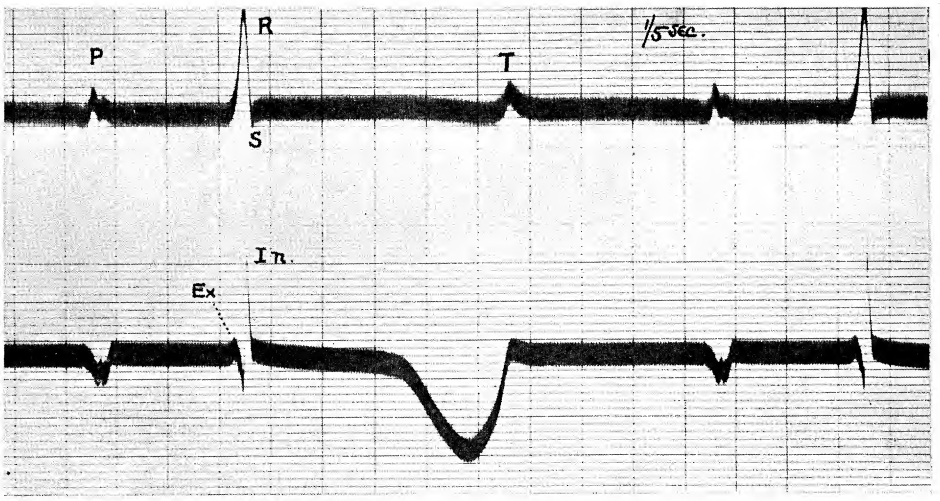


FIG. 7.

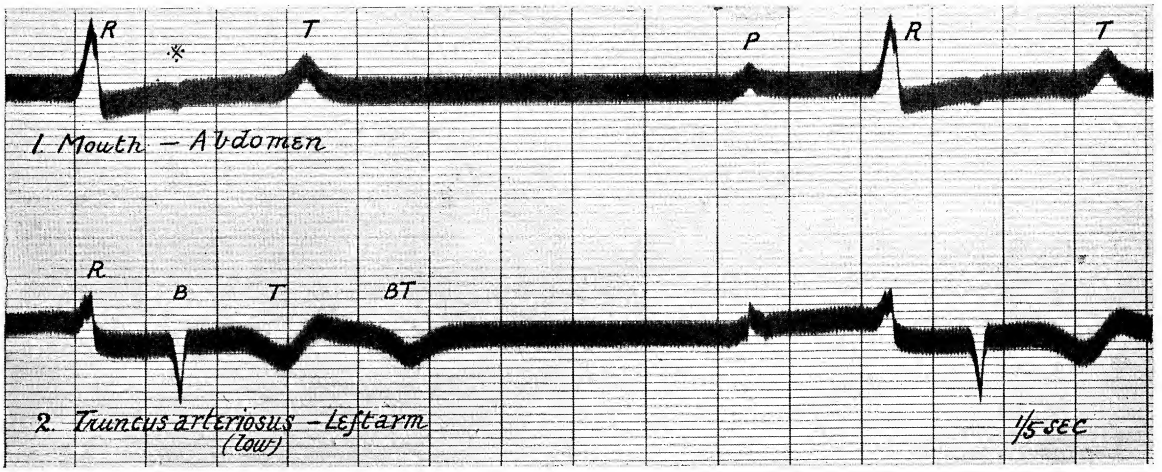


FIG. 8.

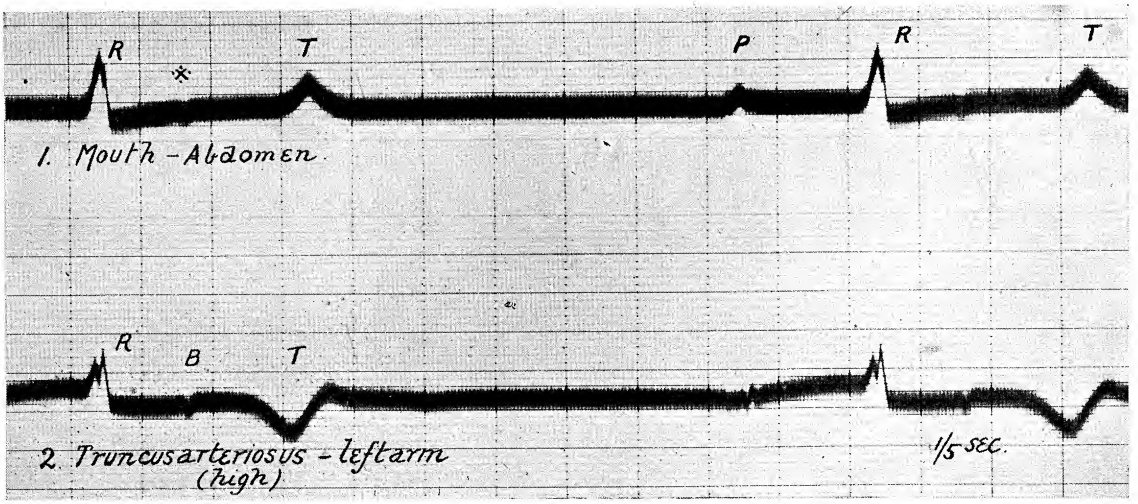


FIG. 9.

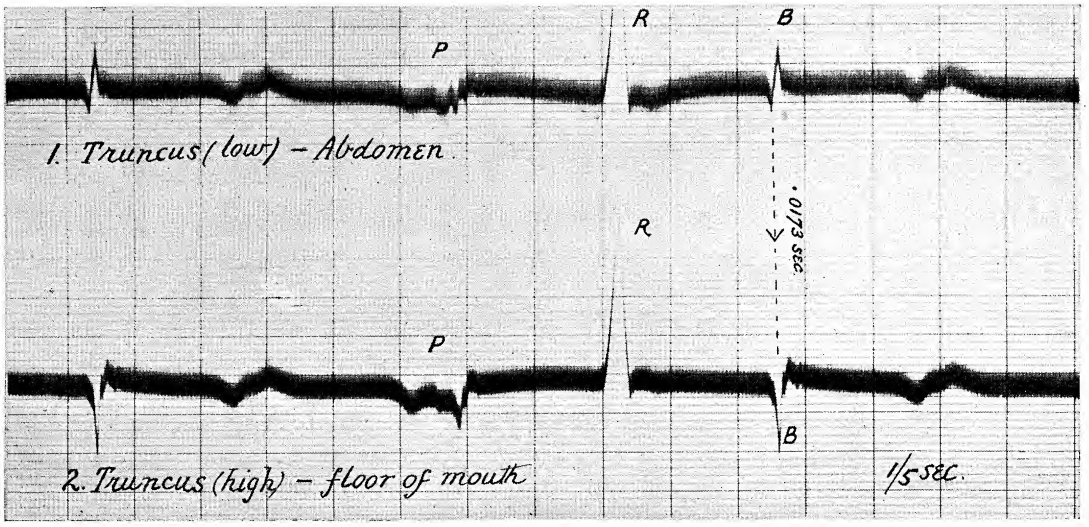

R
B
P
1. Truncus (low) – Abdomen
R
0·173 sec.
P
B
2. Truncus (high) – floor of mouth
1/5 sec.

FIG. 10.

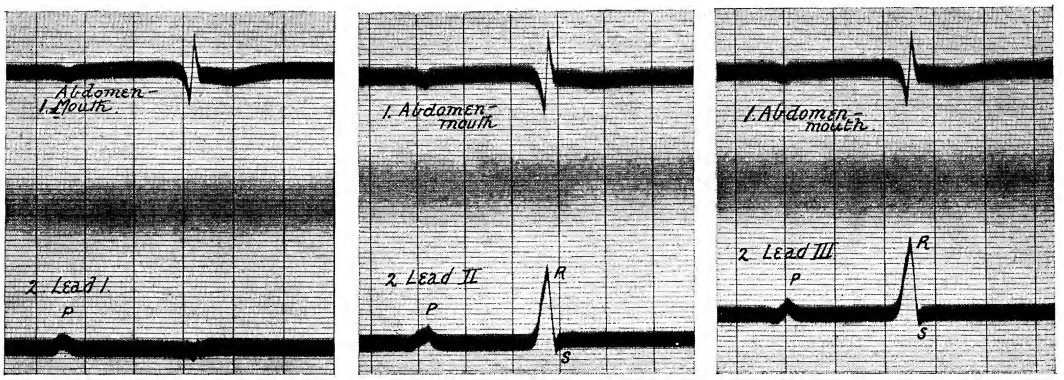

1. Abdomen – Mouth
2. Lead I.
P
1. Abdomen – mouth
2. Lead II.
P
R
S
1. Abdomen – mouth
2. Lead III.
P
R
S

FIG. 11.

EXPLANATION OF PLATES 15 AND 16.

NOTE.—The ordinate values expressed in the explanations of figured curves of this and the succeeding papers are approximate. In taking a curve from any lead for the purpose of charting it, three or more millivolts are introduced into circuit while such a curve is photographed. The extent of the excursion may be found to be a little more or less than the required number of centimetres. This error is eliminated in charting by introducing a corresponding correction.

Fig. 7.—Toad *D*. Simultaneous curves (nat. size): (1) Lead *II*, from right side of pectoral girdle to mid-abdomen; (2) base of ventral surface of ventricle to body wall. The lower curve shows diphasic extrinsic deflections (*Ex.*) followed by a steep intrinsic deflection (*In.*). The reading of the intrinsic deflection is obtained by estimating the interval between the summit of *R* in the upper curve and the commencement of the intrinsic deflection in the lower curve, and subsequently relating it to the commencement of *R*. The vertical lines represent 0·2 second intervals.

Fig. 8.—Simultaneous curves (nat. size). (1) Standard lead from mouth to abdomen; this shows *R* and *T*, the ventricular summits, and a little downward notch marked *, which is due to the passage of the excitation wave through the *truncus arteriosus*. (2) Lead from *truncus arteriosus* to left arm region; this shows extrinsic deflections, which may be recognised as corresponding approximately to *R* and *T*, and intrinsic deflections *B* and *BT*. The *upstroke* of *B* corresponds to the arrival of the excitatory process under the *truncus* contact, *BT* to its subsidence at the same point. The *downstroke* at *B* is an extrinsic effect of the truncus itself.

Fig. 9.—Simultaneous curves from the same animal. (1) The top lead has been maintained. (2) The *truncus* contact has been moved off the musculature of the *truncus* to the commencement of the artery (for actual contact see fig. 4). This curve is consequently different from that of fig. 8. *R* and *T* and another small extrinsic deflection *B* remain, but the intrinsic deflections, *i.e.* the greater part of *B* and the whole of *BT*, have disappeared.

Fig. 10.—Simultaneous curves (nat. size) showing the method of estimating conduction rates. Two contacts are placed on the *truncus arteriosus*, above and below, and each is paired with a separate and indifferent contact on the body wall. The time interval between the intrinsic deflections (first sharp upstroke, *BB*) of the *truncus* is estimated. In this instance it amounted to 0·0173 second. The *truncus* contacts lay 2 mm. apart.

Fig. 11.—Three simultaneous curves ($\frac{2}{3}$ nat. size) from Toad *B*. In each photograph the upper curve is a standard curve from which to measure. The lower curves are taken from Leads *I*, *II*, and *III* (see fig. 3) respectively. The sensitivity of this string was such that 1 centimetre's excursion (10 scale divisions) corresponded to 1 millivolt. Time marker = 0·2 second. These three curves were utilised in constructing fig. 3 and Table V, Toad *B*.

Part II.—*The Tortoise Ventricle.*

[Plate 17.]

Observations have been carried out upon female tortoises of the species *Testudo græca*. They were conducted in the months of May and June, the tortoises being kept in the sun. In an experiment the head was severed from the neck and, the vessels being ligatured, the spinal cord was pithed. The animals were fixed in a sand-bath, the temperature of which was raised. The plastron was trephined in its centre and the cephalic portion removed by diverging saw cuts running forward from the trephine hole.

The leads, arranged around the sides of a triangle, were taken at this stage and before the pericardium was opened. Two contacts were sewn symmetrically over the pectoral girdle, the third was placed on the middle line to the caudal side of the heart. In taking the three leads a separate exocardial lead was used as standard; this lead was from the neck to a point on the caudal and right side of the heart.

Subsequently, the pericardium being opened, direct leads were obtained from the front of the ventricle in the usual fashion, Lead *II* being now employed as the standard.

The distribution of the excitation wave in the tortoise heart in many respects so closely resembles that of the toad that a very full description of it is not considered necessary. This heart serves, however, to illustrate or emphasise important principles.

I find the beat of the tortoise heart, once the pericardium is opened, to be very unstable; it is very sensitive to changes of temperature and may alter appreciably in response to change in the animal's posture. The last change appears to result from the relation of the pericardial fluid to the organ, and may, I believe, be the effect of local temperature changes. My animals have generally been inclined so that the cephalic end of the carapace lay some few inches higher than the caudal end; the pericardial fluid consequently surrounded the apical portions of the ventricle.

In axial exocardial leads the electrocardiogram shows a prominent deflection *R* (Plate 17, fig. 5) and an isoelectric line, or a line which dips as it proceeds, continued into a downwardly directed *T* deflection. As a rule there is no true *S* deflection, but this may be seen in greater or lesser degree, especially before the pericardium is opened. If *S* is prominent then *T* is upright, a relation not without importance, but one which must be left for later and separate research.* These variations are described because the diagrams of surface distribution shown in fig. 1 are, without exception, from hearts showing no *S* deflection and an invert *T*, and it may be open to question whether or not this type represents the most habitual and natural heart-beat in the tortoise. The instability of the tortoise heart clearly emphasises the importance

* There is a similar relation between *S* and *T* in the toad's heart, *T* being upright whenever *S* is prominent and *T* being inverted when *S* is absent. Evidently these facts may help us to recognise the meaning of *T* and its inversion.

of obtaining, as an accompaniment of a series of direct surface readings, standard curves of constant outline, for it is evident to me that any change in the form of the standard curve is accompanied by a change in surface distribution. Observations which bear out this statement deserve illustration, for the fact is of consequence. It will, however, be convenient to deal with this matter more fully at a later stage.

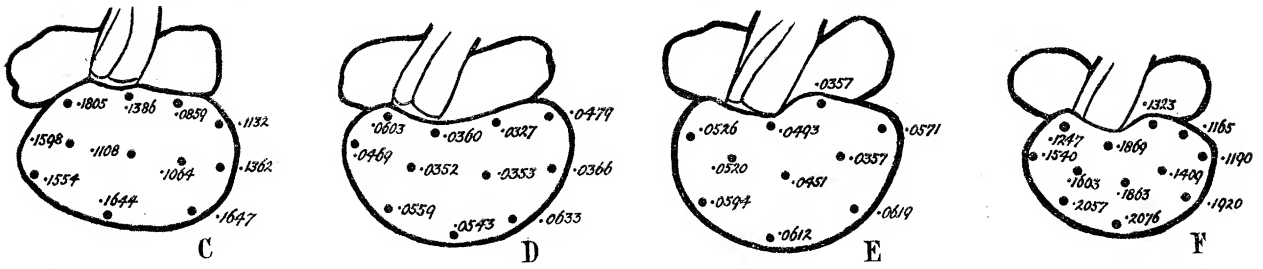


FIG. 1.—Four natural-size outline diagrams to scale of the hearts of Tortoises C–F, showing the surface distribution of the excitation process.

TABLE I.

Tortoise.	Lead *II.*				Surface readings.		Lining readings.			Average length of intrinsic deflections.	Temp.	Heart rate.
	R.	Summit of *R.*	End of *R.*	End of *S.*	Earliest.	Latest.	Near ring.	Farther in.	Farthest in.			
B	0·0000	0·0750	0·1300	None	0·0443	0·0826	0·0102	{0·0362 / 0·0432}	0·0654	0·0250	20–21°	26
C	0·0000	0·1345	0·2137	None	0·0859	0·1805	—	—	—	0·0155	21–22°	33
D	0·0000	0·0546	0·0932	None	0·0327	0·0633	0·0145	0·0314	—	0·0158	29–30°	39
E	0·0000	0·0593	0·1028	None	0·0357	0·0619	0·0144	0·0269	0·0600	0·0234	27°	52
F	0·0000	0·1446	0·2732	None	0·1165	0·2076	—	—	—	0·0293	20°	24
G	0·0000	0·0496	0·0900	None	0·0319	0·0544	0·0115	—	0·0387	0·0177	21–30°	32

When the pericardium has been opened, the distribution of the excitatory process over the ventral surface is as illustrated by the outline diagrams of fig. 1. It is not dissimilar to that found in the toad. It is the rule that the apical portion of the ventricle is latest, the base and central region, or the central region, earliest. From time to time a high reading is obtained from the region of the base in the neighbourhood of the *truncus arteriosus*. The right margin is sometimes activated at a later time than the left margin. The whole ventral surface is activated in two or three hundredths of a second (fig. 1, Tortoise *D*, *E*) when the initial phases of the axial electrocardiogram are relatively short (Table I) and the heart beat active. The completion of surface activation takes longer when the initial phases are of greater duration (Tortoise *C*, *F*). The arrangement and values of the readings, compared with the surface conduction rates in the same hearts (Table II) preclude the idea of a simple surface spread and suggest, as in the toad, a distribution from within outwards. As in the toad's heart the time period of the initial phases is only partially covered by the surface readings. Thus in Tortoise *B* (fig. 2) the initial phases of the three leads shown in the corresponding diagram have a total duration

TABLE II.—Rates of Conduction.

Tortoise and temperature	Interval.	Distance.	Rate	Direction.	Distance of stimulating electrodes.
	secs	mm.	mm. per sec.		mm.
B (20–21°) . .	0·0420	7	169	↑	—
B	0·0447	7·3	163	→	3
	0·0467	7·3	156	→	12
B	0·0566	8·5	150	←	5
	0·0567	8·5	150	←	14
C (21–22°) . .	0·0987	9	91	←	7
C	0·0807	10·5	130	↖	6
C	0·0753	7	93	↗	3
	0·0739	7	95	↗	11
D (29–30°) . .	0·0308	6	195	↖	4
D	0·0229	4	175	↗	11
E (27°). . . .	0·0256	7	273	↗	4
E	0·0291	6	206	↗	4
E	0·0241	6	249	↗	10

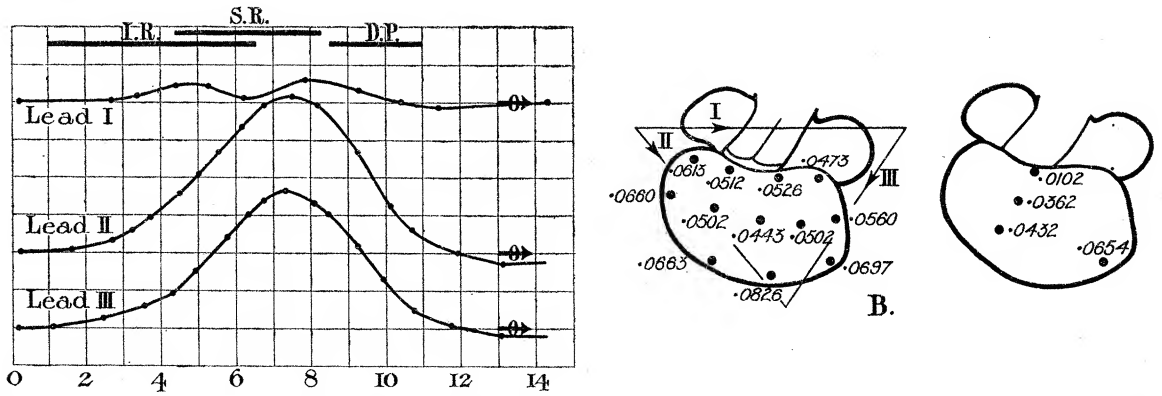


FIG. 2 (Tortoise B).—A chart showing the initial phases of the electrocardiogram in Leads *I*, *II*, and *III*. Ordinates, 3 cm. (6 scale divisions) = 1 millivolt; abscissæ, 1 cm. (2 scale divisions) = 0·02 second. The left-hand diagram shows the surface readings for the same heart and the relation of the three leads of the chart to the ventricle in the same animal. The right-hand diagram shows four readings from the lining of the same ventricle, taken approximately from the points shown. All the readings are related to the 0 line of the chart. *I.R.* = period covered by internal readings; *S.R.* = period covered by surface readings; *D.P.* = period covered by developing potential difference.

of some twelve or thirteen hundredths of a second, the superficial readings for the ventral surface corresponding to the period covered by the line *S.R.* They correspond to the last half of the rise of *R* and to its summit. There is, as in the toad, an initial and a terminal hiatus. The terminal hiatus is to be explained chiefly by the time taken in the full development of the charge, which amounted, as estimated from the length of the intrinsic deflections, to at least 0·0250 second in this animal (fig. 2, line *D.P.*). The initial hiatus is to be accounted for by the early activation of the muscle, which lay deeply. The tortoise heart is more manageable than that of the toad, and the positions of internal contacts are more readily determined. The earliest readings are taken from that portion of the lining which borders the *A-V* ring and, as the contact is moved towards the apex of the ventricle, so the readings are later. In fig. 2 a second diagram, that to the right, shows the positions of four internal contacts and the corresponding readings for Tortoise *B*. They cover practically the whole of the initial hiatus and overlap the surface readings to a considerable extent (fig. 2, line *I.R.*). The earliest readings obtained lay 0·0102 second after the earliest trace of *R*, the latest reading some 0·0654 second after the same phase of the initial deflections in the axial electrocardiogram. It is clear from a diagram of this kind that the initial phases correspond to the activation of the ventricle as a whole ; a rule which applies to all vertebrate hearts, as we shall eventually discover. In the tortoise, the rule is further illustrated by Table I, where earliest and latest readings are given side by side with the length of the initial phases of axial curves; absolute correspondence is not to be anticipated, for the whole surface and lining is not investigated in a given animal. But the Table shows an unmistakable relation between the length of the initial phases and the duration of the activation process, as estimated from direct readings. Briefly, the axial curve in its initial phases is a summation of those electrical effects which signal the involvement of the ventricle in all its parts.

Thus the duration of the initial phases is controlled by the speed with which the excitation wave is distributed, and in beats of natural type the speed is mainly governed by the conducting power of the muscle elements. In support of the latter statement it may be noted that where the estimated conduction rate approaches 200 mm. per second (Table II, Tortoise *D* and *E*) the initial phases have an approximate duration of a tenth of a second. Where the conduction rate approaches 100 mm. per second (Table II, Tortoise *C*) the initial phases last approximately two-tenths of a second ; where, as in Tortoise *B*, the conduction is of intermediate rate, the length of the initial phases is also intermediate.

As the duration of the initial phases is controlled by the speed of distribution, so the form of these phases is controlled by the paths of distribution. The direct readings convey an imperfect idea of these paths, though indicating a general resemblance to those of the toad's heart. The electrical axis and its movement complete our information. It is unnecessary for our purpose to calculate the exact

electrical axis for the heart of the tortoise; for as the chief differences in potential are found between the contacts forming Leads *II* and *III*, the curves of these leads being of considerable magnitude and almost identical in outline, while the curve of Lead *I* is always diminutive, so we may conclude that the electrical axis is almost in the axis of the body throughout the whole of the initial phases. And, the chief of these phases being upward, the angle formed with our base line is constantly in the neighbourhood of 90°. It may be that it turns abruptly to −90°, as in the frog, when an *S* develops; but in most of the studied instances a straight base-apex spread has been maintained. In a solitary and peculiarly instructive experiment this mode of spread was temporarily in abeyance. This experiment is illustrated by fig. 3. The curves of the triangular leads are charted, the distribution on the surface

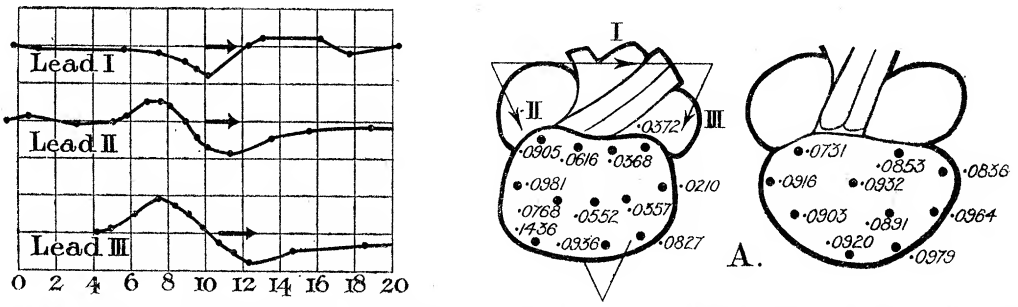


FIG. 3 (Tortoise A).—A chart showing the initial phases of the electrocardiogram in Leads *I*, *II*, and *III*. Ordinates, 3 cm. (6 scale divisions) = 1 millivolt; abscissæ, 1 cm. (2 scale divisions) = 0·04 second. The left-hand diagram shows the surface readings corresponding to the chart and the leads employed in obtaining the curves of the chart. The right-hand diagram shows a later and more normal surface distribution of the excitatory process in the same animal, when the curves of the triangular leads had resumed their customary outlines.

TABLE III.—Tortoise A.

Second.	*I.*	*II.*	*III.*	Angle.
0·0400	−1	−1	0	↑ −150°
0·0500	−1	0	1	↑ 150
0·0600	−1	2·5	4 (3·5)	106
0·0700	−2	5	8 (7)	106
0·0800	−3	5	8	112
0·0900	−5	0	5	150
0·1000	−7	−7	0	−150
0·1100	−5	−8	−4	−124
0 1200	−1	−8	−7	− 97
0·1300	2	−6	−8	− 76
0·1400	2	−4	−6	− 71
0·1500	2	−3	−5	− 67
0·1600	2	−2	−4	− 60

is shown in the left-hand diagram. Now the spread on the surface is unusual in several respects. First, it is unusually slow; second, the left margin is

supplied earlier than any other region of the ventral surface; and third, the readings show an orderly sequence from left to right. Unquestionably, this mode of spread is unnatural; it was observed in the first tortoise examined, and before the influence of temperature had been fully recognised (the actual temperature of the beast was 18°). I imagine that the impulse was impeded in its spread through the A–V ring, being confirmed in this supposition by finding an unusually prolonged P–R interval in the associated curves, and that the impulse spread to the ventricle entirely or chiefly through the left sector of the ring. From this margin it would follow the musculature, and proceed across the ventricle from left to right, down the left margin and along the broad apex from left to right, to rise finally, perhaps, on the posterior surface and extreme right margin. When we examine the charted curves and the calculated electrical axis (Table III) we discover corresponding features. The initial phases are exceptionally long. At the very beginning, the angles are of doubtful value and sign, but from 0·0600 second* onwards, they are trustworthy. The path of spread which is indicated is at first from above downwards and slightly from left to right (106° to the horizontal); it remains at this angle for a while and subsequently changes in a regular clockwise fashion through 194°, the predominating direction being from left to right. The general spread, so far as the whole musculature was concerned, was at first from base to apex, later from left to right, and finally from below upwards. Whether a local conduction defect in the ring is a sufficient explanation of this unusual distribution need not detain us; I use the experiment to illustrate the close connection between the order of the direct readings and the direction and change of the electrical axis. And this experiment is of greater value, because, maintaining the animal in a warm atmosphere, the usual type of curves from extracardial leads subsequently prevailed.† A second series of surface readings was obtained, and is shown in the right-hand outline diagram of fig. 3. The general arrangement of these later values was the usual arrangement for the tortoise.‡

The investigation of the tortoise ventricle produces no evidence that there is in this organ any special tissue which conducts more rapidly than the surface muscle fibre. In this respect the heart is like the toad's, and my reasons for this statement are similar to those enumerated for the toad's heart. There is the additional argument, in the case of the tortoise heart, that in estimating the rate of conduction, the distance of the stimulating electrodes from the proximal contact is without

* The earliest surface reading is 0·0210 second; allowing 0·0250 second for the development of the discharge, we arrive at the figure 0·0461 second.

† These curves were of the type shown in fig. 2, but the heart being still a little unstable when the three leads were taken, and the series failing as a consequence to show a perfect fit, I do not reproduce a corresponding chart.

‡ The values of this diagram are perhaps not strictly compatible with each other, because the heart beat was still somewhat unstable; however, it was sufficiently stable to render them compatible in a general sense.

influence upon the result. In the dog's heart, if the stimulating electrodes are at a distance, the excitation wave has time to traverse the thickness of the muscle wall, and entering the Purkinje arborisation is carried rapidly to the two contacts between which the conduction rate is tested. Removing the stimulating electrodes from a point near to, to a point far from, the proximal contact, results in a notable increase of conduction rate between the two contacts in this animal. In the tortoise the conduction rate is unaltered by this procedure (Table II), and I conclude from this and the remaining arguments (see Part I) that a system having special powers of conduction does not exist in this animal.

The chief difference between the distribution in the toad's heart and in those tortoise hearts which I have examined is to be found in the relatively early activation of the base of the ventricle in the latter. This feature is entirely compatible with the absence of an *S* deflection in the extracardial axial leads of the corresponding animals, and confirms the view that *S*, appearing in an axial lead from the toad's heart, is due to the late activation of basal muscle.

In the tortoise ventricle the fusion of the ring musculature with the ventricular wall at a level relatively nearer to the base than pertains in the toad's heart seems a sufficient explanation of this minor difference in the mode of spread in the two beasts.

EXPLANATION OF PLATE 17.

Figs. 4, 5, and 6 (Tortoise *G*).—In each of these photographs the upper curve is the standard curve from the neck to a point outside the right and caudal aspect of the ventricle, the lower curves are from the leads *I*, *II*, and *III* of the triangle.

Ordinates of lower curves, 1 cm. = 1 millivolt; abscissæ = 0·2 second.

Figs. 7, 8, and 9 (Tortoise *G*).—Fig. 7: A direct lead from the lining of the ventricle near the *A–V* ring and an indifferent point on the body wall, taken simultaneously with Lead *II*. Fig. 8: Similar curves, the direct lead being from the ventricular surface near its centre. Fig. 9: Similar curves, the direct lead being from the lining of the heart near the ventricular apex.

In each direct lead an extrinsic deflection (*ext.*) and an intrinsic deflection (*int.*) is shown. The extrinsic deflection is very deep in fig. 9, but the intrinsic deflection rises from it sharply.

Ordinates of direct curves, 1 mm. = 1 millivolt; of lower curves, approximately, 1 cm. = 1 millivolt; abscissæ, 0·2 second.

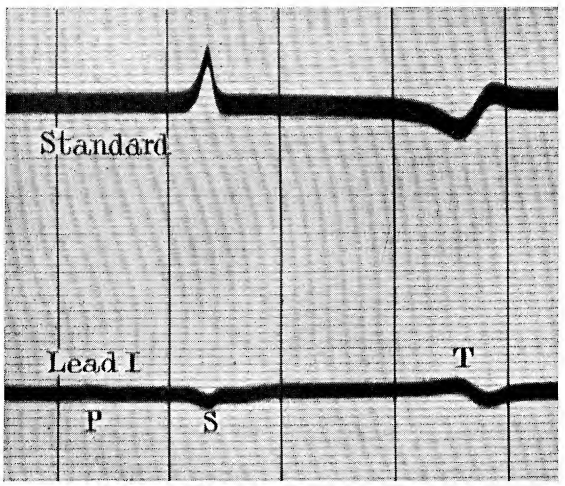


FIG. 4.

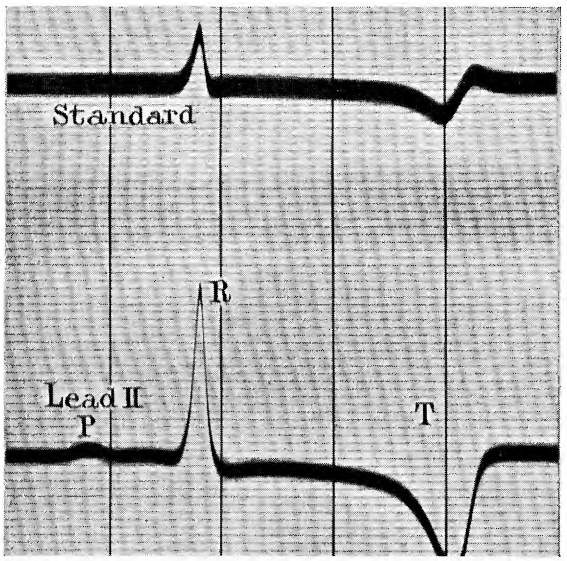


FIG. 5.

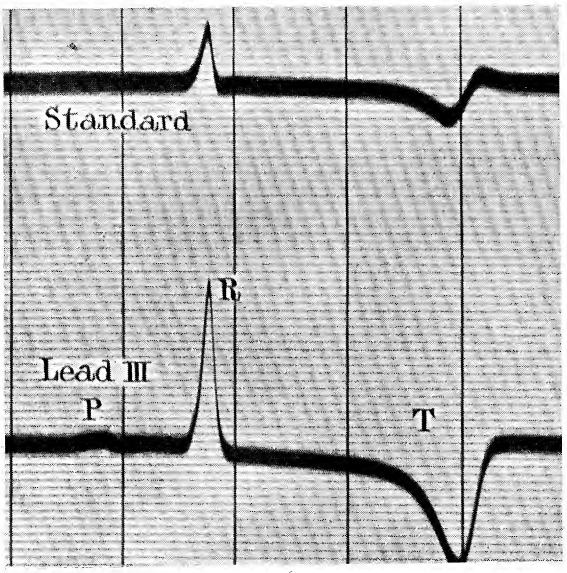


FIG. 6.

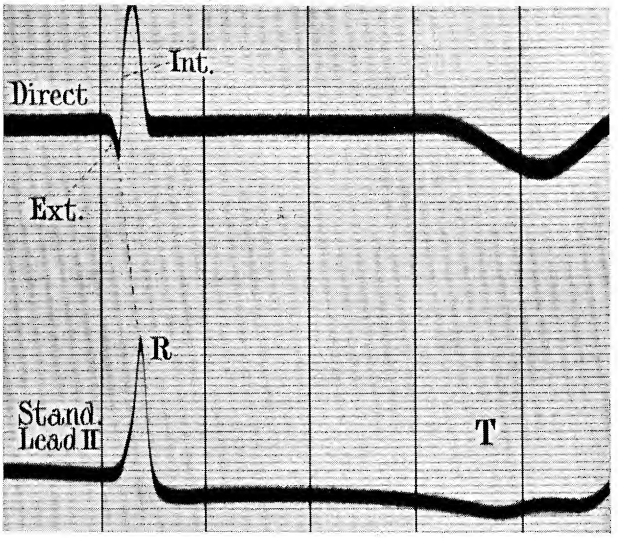


FIG. 7.

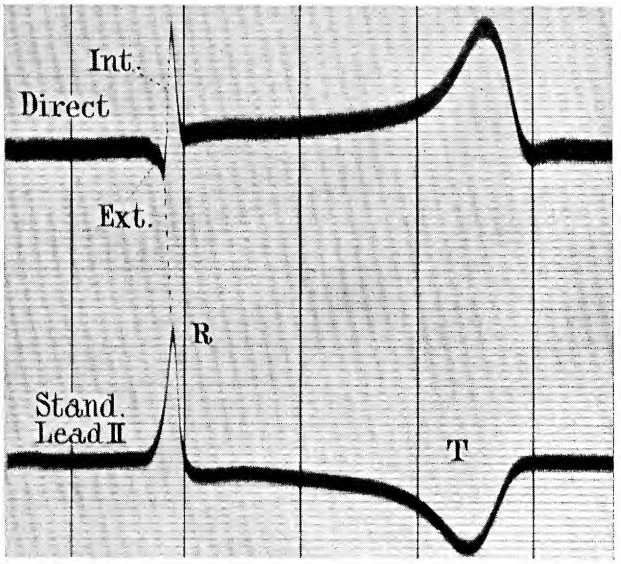


FIG. 8.

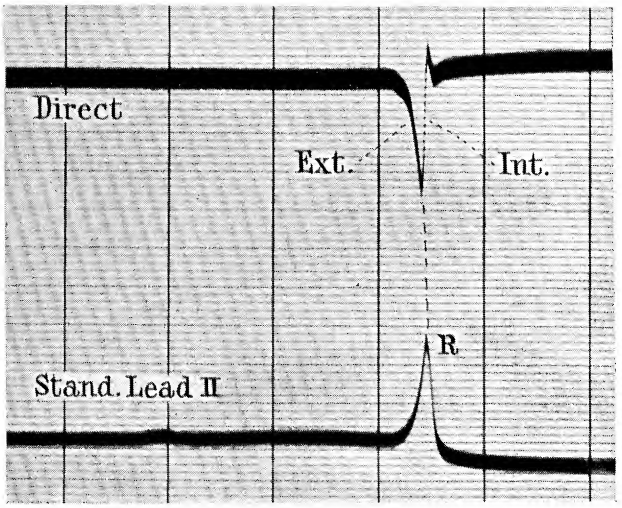


FIG. 9.

Part III.—*The Dog's Ventricle.*

[PLATES 18–20.]

THE DUALITY OF THE VENTRICULAR ELECTROCARDIOGRAM.*

In recent observations upon the dog's ventricle ROTHSCHILD and I advanced experimental evidence which we believe shows conclusively that the excitation wave is distributed to the mammalian ventricle through the arborisation of the auriculo-ventricular bundle and its branches.† In our paper we published a number of maps exemplifying the distribution of the excitatory process over the surface of the ventricles in the dog. The readings of commencing surface activity, as there portrayed, are related to one another in a manner which must now receive emphasis. So far as the right ventricle is concerned, the earliest sign of activity appears over what we term the central region of the ventral surface, a region situate close to the ventral attachment of free wall to septum. As the readings are traced from this region towards the auriculo-ventricular groove, they become gradually later in point of time; they show a similar increase in value as they are traced towards the actual attachment of the septum. Over the whole of the right ventricle, neighbouring readings either approximate to one another very closely or show a gradual succession. The last statement applies precisely to the surface of the left ventricle also. But the statement does not apply to the ventricles treated together. Speaking especially of the ventral surface, the readings on one or other side of the septum diverge notably from each other, the higher readings being over the left ventricle. This is no doubt in great part due to the greater thickness of muscle on the left side and to the consequent delay in the arrival of the excitation wave at its surface; but the relatively abrupt change is evidently conditioned also by the isolation of the spread to the free walls of the right and left ventricles. That is to say, the whole free wall of the right ventricle receives its supply from the right bundle division, the whole free wall of the left ventricle is supplied by the left bundle division; during the natural heart beat the excitation wave does not spread from the border of one ventricle to the neighbouring border of the other ventricle. The two waves spreading on right and left sides meet and end in the septum. This conclusion, which is alone compatible with the normal surface distribution whose study first prompted it, is completely borne out by comparing the surface readings before and after section of a single large branch of the distributing tracts on right or left side. We showed in our paper that if the large septal branch of the right division,

* A preliminary report of these observations appeared in the 'Proceedings of the Physiological Society,' March 13, 1915.

† 'Phil. Trans.,' B, vol. 206, p. 181 (1915).

which supplies at least the whole ventral surface of the right ventricle, is divided, the surface readings over the left ventricle remain unaltered relative to each other; but that the readings over the ventral surface of the right ventricle are all disturbed, being later in point of time and of such values as to show a spread of the excitation wave from left ventricle to right. Briefly, the described procedure abolishes that feature of the physiological heart beat which is so noteworthy, namely, the strict confinement of spread through a given bundle division to the corresponding ventricle.

During the course of the present experiments these observations have been repeated and extended; and it may now be stated in more general terms that if one or other of the two chief divisions of the A–V bundle is interrupted, the spread to the contralateral ventricle is unaffected, while the spread over the homolateral chamber is profoundly altered; the direction of travel subsequent to the injury being from the side contralateral to the lesion towards and over the side homolateral to the lesion.

In view of these observations I conclude that the normal axial electrocardiogram is in reality a *bigram*, a dual picture of the curves of right and left ventricle respectively; the *bigram* consists, according to this hypothesis, of the algebraically summated *dextrogram* and *levogram*.

My next endeavour has been to bring forward proof of this proposition, and the following experiments have been directed to that end.

We may pause for a moment to consider the curves obtained by interrupting the right or left bundle division, respectively. A lesion of the right stem produces in Lead *II*, a curve of the form shown in the central curve of Plate 18, fig. 12.* The change from the natural type (Plate 18, bottom curve of fig. 12) is conspicuous and characteristic. We may term such curves *levograms*, with a single qualification. They are true levograms in their initial phases only. While the initial phases are being inscribed the excitation wave is spreading, as observation upon surface distribution clearly shows, along perfectly natural paths in the left ventricle, while the whole right ventricle remains inactive. But after a time interval of 0·0350 to 0·0400 sec. has elapsed,† the spread is complete throughout the left ventricle, and has commenced its abnormal course through the right ventricle; from this point onwards therefore the curve is no longer a true levogram.

Precisely similar arguments apply to the curves yielded by lesions of the left bundle division (Plate 18, top curve of fig. 12). In their initial phases they are true expressions of the dextrogram. By using a special form of clamp‡ it is possible to obtain in one and the same animal the curves corresponding to right and left

* That the curves correspond to the lesions named is for the moment partly assumed; the evidence will be given in detail at a later stage of this report.

† From the commencement of R.

‡ The clamp adopted has been described by MEAKINS, the curve of the instrument being modified slightly to throw its jaws more towards the ventral surface of the heart.

bundle branch lesions, by alternately compressing these branches. The experiment is not easy to perform, and is precarious to this extent, that it is not possible subsequently to identify the tissue areas involved in the compression. Gentle pressure is exerted, working from the ventral towards the dorsal border of the septum at its base, until abruptly the normal axial curve vanishes, and is replaced by a curve which is recognised as characteristic of a right branch lesion. The necessary observations upon this disturbance being completed, pressure is relaxed and recovery is awaited; when the normal curve is resumed—the recovery is gradual—the forceps are carried a little more dorsalwards, and the compression re-applied. In a successful experiment, a curve characteristic of a left branch lesion results.

Thus three sets of curves are obtained: the normal, and those which include the initial phases of the true *levogram* and *dextrogram*. All curves are standardised, so that 3 cm. of excursion are equivalent to 3 millivolts of potential.

The next step is the orientation of these curves so that they may be charted with corresponding phases in time lying vertically above each other. The procedure is as follows: the ventral surface of the heart being exposed, the natural curve from Lead *II* is used as a standard of measurement; and, simultaneously with this, successive curves are taken by means of direct leads* from suitable points on the surface of the right and left ventricle (see contact points in fig. 1). The present illustration is one of two examples.

The times at which the excitation wave appears at these points, relative to *R* in the standard lead, are estimated (these readings are placed immediately below the contact points in fig. 1).

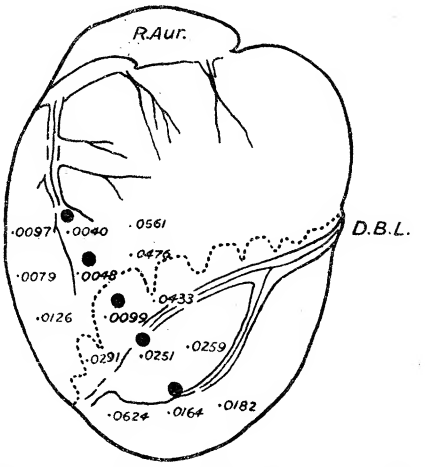


FIG. 1.—Outline drawing to scale (2/3 natural size) of the heart of Dog H.H. *D.B.L.* = descending branch of left coronary artery. Five contact points were used in this instance, curves being taken from each separately paired with a constant chest wall contact and simultaneously (*a*) with the natural electrocardiogram from Lead *II* (the corresponding readings for the surface points are placed immediately below the latter), (*b*) with the levogram (the corresponding readings are placed to the right), and (*c*) with the dextrogram (the corresponding readings are placed to the left). The dotted line represents the right attachment of the septum.

The clamp is now applied and the curve corresponding to a right branch lesion obtained; fresh readings are then taken from the original contact points, using the small initial upstroke of the levogram as standard (these readings are placed to the right of contact points in fig. 1). After recovery a third series of readings is taken, using the upstroke of the dextrogram as standard (these readings are placed to the left of the contact points in fig. 1).

* The direct lead is from a contact with the surface of the heart and an indifferent point on the chest wall, as hitherto adopted.

Now if the readings of surface points for the left-sided beat are compared with those of the natural beat, it will be seen that the order over the *left* ventricle is unchanged, and that the actual values change only slightly. The same statement applies to the surface of the right ventricle when the natural and right-sided beat are compared. Over the ventricle homolateral to the lesion the values are greatly raised and are arranged in a fashion indicating spread from the undisturbed side.* The slight change in the actual readings over the contralateral surface is due to alteration of the standard of measurement, for it varies in the same direction and by approximately the same amount in each instance.

In charting, I take the beginning of *R* in the natural curve of Lead *II* and place it on the 0 time line (fig. 2), subsequently plotting the whole of the initial phases of the bigram. In charting the initial phases of the levogram, I commence the small initial upstroke of this curve by plotting it relative to *R*; this point in time is arrived at by averaging the differences in the two sets of corresponding readings over the left ventricle. Thus *R* is placed on the 0 time line for the heart of fig. 1; the differences, 0·0259—0·0251 = 0·0008, and 0·0182—0·0164 = 0·0018, give an average of 0·0013. The initial upstroke of the levogram is charted as preceding the upstroke of *R* in the natural curve by 0·0013 second (see fig. 2).

Similarly the average difference between the two sets of readings over the right ventricular surface, corresponding to natural and right-sided beats, is used in charting the dextrogram. Thus, 0·0097—0·0040 = 0·0057, 0·0079—0·0048 = 0·0031, and 0·0126—0·0099 = 0·0027, the average difference being 0·0038; the upstroke of the dextrogram is charted as commencing before *R* by this time interval (see fig. 2).

Thus I am able to chart from one animal standard curves corresponding to the natural beat (fig. 2, *N*), the left-sided beat (fig. 2, *Lf*), and to the right-sided beat (fig. 2, *Rt*), and to place them with corresponding time phases on the same vertical line. If now the left- and right-sided beats are summated algebraically, a calculated curve is obtained (fig. 2, *C*). This curve is to all intents and purposes a duplicate of the natural curve both in respect of its time relations and in respect of the voltages expressed by its different deflections. Even the notch on the upstroke of *R* is clearly and accurately repeated in the illustration. It departs from the form of the natural curve where it may be expected to depart from it, namely, at 0·0350—0·0400, for after this time interval the left- or right-sided curves no longer represent pure levogram or pure dextrogram.

Thus it is shown that in its initial phases the natural electrocardiogram is a composite curve, built up of the superimposed effects of right and left ventricle

* The unaltered relation of the readings on the side unaffected by the lesion and their characteristic increase over the affected side is a sufficient check upon the nature of the curves obtained. It is not only upon the appearance of curves of characteristic shape, but upon these readings, that I base the assumption that conduction in the bundle branches has been successfully and alternately hindered in a given experiment of the kind now described.

respectively. It is a *bigram* comprising an algebraically summated *dextrogram* and *levogram*.

This initial step in the analysis of the axial electrocardiogram is important. Clearly, the further analysis should consist in separate analyses of dextrogram and levogram respectively.

Meanwhile we may point to the origin of certain deflections in the bigram in terms of left or right ventricle. *Q* appears, so far as these observations go, to arise in the dog from the left ventricle in Lead *II*. Where *Q* is absent in the bigram it is absent in the levogram. *R* is in the main a right-sided event, but may be added to by the curious and almost constant hump (marked by an asterisk in fig. 2) of the levogram. *S* is decidedly the result of the preponderating downstroke of the levogram.

The analysis is also valuable in explaining variations in the form of the natural curve. Evidently slight variations in the time relations of dextrogram and levogram will profoundly modify it. Thus the hump of the levogram, falling early, notches and broadens the upstroke of *R*; falling later it blends with and raises the summit of *R*. Of other possible variations I shall speak more fully at a later stage.

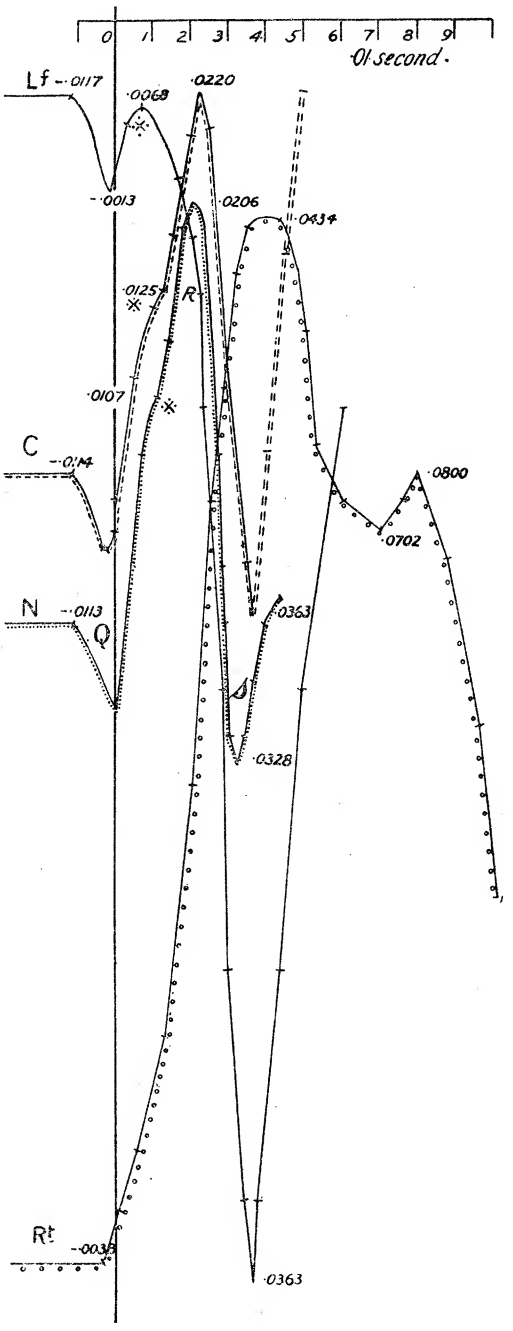


FIG. 2.—A chart of four curves. *Lf* is the levogram, *C* is the calculated bigram, *N* is the actual bigram, and *Rt* is the dextrogram of Dog H.H. All the curves are plotted in relation to *R* of the natural bigram, and this stands on the 0 line. The calculated bigram (*C*) has been constructed by algebraic addition of the values found on corresponding vertical lines in dextrogram and levogram. It is an almost exact replica of the actual bigram, both in respect of its time relations and in respect of the voltages represented by the several deflections. Note especially the notches marked by asterisks on the upstrokes of *R*. The calculated curve departs from the outline of the natural curve approximately some 0·0350 sec. after *R* begins, or approximately 0·0450 sec. after the first phase of the levogram. Ordinates are on the scale of 7·5 cm. = 1 millivolt. Abscissæ, 1 cm. = 0·02 sec.

There are two possible criticisms of these experiments. It may be argued that

in clamping the right division certain branches of the left division are also injured. The reply to this criticism is to be found in protocols of experiments published later (p. 267); unless there is very extensive damage or complete transection of the left division the distribution to the left ventricle as gauged by the extracardial leads remains unaltered. The second criticism which may be raised is that in several applications of the clamp the main stem of the bundle is almost certain to suffer some damage. As a matter of fact, in one series of my curves 2 : 1 block was present; evidently in this circumstance the impulse was still of auricular origin, and the spread was therefore still through the usual Purkinje channels. I possess similar curves from this animal in which there was no block, but the curves, being tremulous, were less suitable for measurement, though of the same outline. Criticism applies chiefly to the other experiment in which complete heart-block was present (fig. 2, and Plate 18, fig. 12). The reply to this criticism is also that the curves are of the same form whether complete block is present or not. An experiment performed for the purpose is illustrated by Plate 18, figs. 13 and 14. In this animal a clean cut severed the right division of the bundle, and the usual curves were subsequently recorded (fig. 13); a clamp was now placed on the bundle stem and tightened until complete block was obtained (fig. 14). Despite the dissociation of auricular and ventricular rhythms, the form of ventricular complex remained practically unchanged. Such slight change as occurred is no more than is observed from time to time in an experiment in which the curves are repeatedly taken without further experimental interference. The uniformity of distribution to a given ventricle, when the contralateral bundle division is not conducting, before and after the production of auriculoventricular dissociation, is readily to be explained upon the usual assumption that in $A-V$ dissociation the impulses spring from the bundle itself immediately below the seat of damage. As a matter of fact, full justification of the primary experiment lies in its end result: the algebraic relation between dextrogram, levogram and bigram. Such accurate fitting cannot be coincidental and can be explained only by the hypothesis suggested.

SEPARATE CONSIDERATION OF THE LEVOGRAM AND DEXTROGRAM.

We owe to EPPINGER and ROTHBERGER* the statement that lesions of one or other division of the $A-V$ bundle produce profound modifications of the natural electrocardiogram in dogs. Their general statement and their account of the form of change has been accepted widely by a number of workers upon the physiology and pathology of the heart. I have accepted their general conclusions, and have had less hesitation in so doing because they have seemed to me to harmonise with a variety of physiological and clinical observations. At the same time, it should be stated that the report in question and the subsequent article of ROTHBERGER and

* 'Zeitschr. f. klin. Med.,' vol. 70, p. 1 (1910).

Winterberg* do not contain a sufficiently detailed statement of procedure and evidences to ensure, by themselves, unqualified support to the conclusions contained in them. Eppinger and Rothberger's experiments have not as yet been confirmed by successful repetition.

As the conclusions of my present article must presume those of Eppinger and Rothberger's to be valid, it has been necessary to seek corroboration of their observations. The general methods employed have been sufficiently recorded in previous articles. The curves have been studied in three leads from pairs of non-polarisable electrodes fixed to the right shoulder, left shoulder, and the mid-line of the abdomen, immediately above the umbilicus. A standardised curve has been taken from each lead (each simultaneously with a constant curve from a direct heart lead), before and after the insertion of the knife. Extra sets of curves have been taken whenever the change in the form of curves has been unstable. The extent of the lesion has been subsequently examined.

The Change in the Electrocardiogram Produced by Lesions of the Right Bundle Division of the Dog.

Proof of the Association of the Lesion with a Particular Type of Electrocardiogram.

The right stem of the A–V bundle in the dog, after leaving the bifurcation, runs in the septum on its right side first in a ventral direction and in almost the whole of its course it is covered by endocardium and a connective tissue sheath only. In the majority of freshly opened hearts, its course can be faintly but distinctly traced with the naked eye. The branch curves, being convex toward the ventral surface of the heart, and finally enwraps the chief papillary muscle of the right ventricle. This course is displayed in the accompanying outline drawings (fig. 3).

These diagrams have been constructed accurately to scale, and with the septum displayed in a constant fashion; the free wall of the ventricle is detached by a cut running from the pulmonary orifice along the A–V groove, and finally ascending into the auricular wall; the freed ventricular wall (*F. W.*) is thrown downwards, and the attachments of those chordæ which go to the chief papillary muscle (P) are divided.

In the upper part of its course the bundle division (B) is hidden by the septal segment of the tricuspid valve (*S. T.*). When it reaches the papillary muscle it breaks up into a number of free and anastomosing branches. Other branches are frequently given off in the lower part of the convexity; and these may be visible (see fig. 3, *II*, *IK*); but more often they are found by microscopic examination. The dotted lines show that portion of the course of the bundle division which was visible in the fresh heart.

In my early experiments on the right bundle division I used a clamp, and, having lightly pressed with this instrument until the desired curves were obtained, increased

* 'Zentralb. f. Herz- u. Gefässkrank.,' vol. 5, p. 206 (1913).

the pressure and obtained a permanent effect. In a series of seven successful experiments on the dog, discoloration of the tissues or blood clot was found on the right side of septum, clearly overlying the right bundle stem; but it was also apparent in many of these experiments that some of the ventral branches of the left division were also involved. I cut three of the dog's hearts in serial sections. In one dog the region of crush could not be positively identified microscopically. In the other two the right division suffered chief damage, but the bifurcation of the bundle was also involved in spreading hæmorrhage.

These experiments were regarded as indecisive, and the method of clamping was therefore abandoned.

In dividing the right division of the bundle I introduce a tenotomy knife through the centre of the conus muscle, immediately beneath the pulmonary valves; the blade is passed into the cavity on the flat and parallel to the $A-V$ groove. Once through the wall, the point is carried a little towards the heart's apex to escape entanglement in the chordæ tendineæ, and the cutting edge is turned toward the septum. The knife is then pushed on until its point is actually in line with the base of the right auricular appendix, or a little beyond it. The cutting edge is applied to the septum, the cut made, and the knife withdrawn. Little or no hæmorrhage accompanies this operation, and a successful cut may be placed in at least 50 per cent. of experiments.

The figure illustrates eight experiments. Preliminary curves were always taken from the three leads, and extra sets during the course of the experiment. Each heart was examined by means of serial sections.

Dog I.J. (5·5 kgrm.). A cut was made at the ventral edge of and below the septal valve. The bundle division could be traced to the centre of it in the freshly opened heart and microscopically. The bundle was completely divided at the centre of the incision. Characteristic curves (fig. 17) at once developed, and these were maintained permanently.

Dog I.L. (5·0 kgrm.). Two cuts which fell together, and the first of which was ineffectual, were made. In this animal the course of the bundle division could be traced for a short distance only. It seemed to the eye to have been involved in the tails of the incision. The sections showed the bundle division to be completely transected by the upper of the two incisions at its extremity. Curves of small amplitude, but of characteristic form, developed at once and were maintained.

Dog I.M. (11·3 kgrm.). A single cut was made, into the edges of which the bundle division could be clearly traced. The sections showed transection of the bundle division. Characteristic curves at once developed and were permanently maintained.

Dog I.I. (9·3 kgrm.). The preliminary and physiological curves were of a somewhat unusual type. A cut was made below the septal valve. The bundle branch was readily traced into the middle of it, and sections showed it completely divided. The electrocardiograms at once changed. In Lead *II* the curve was of the usual and characteristic form; in Lead *I* it was of similar form and of equal extent; in Lead *III* it was anomalous.

Probably the atypical characters in Leads *I* and *III* were associated with an unusual distribution of the Purkinje tissues to the left ventricle; here the free strands were exceptionally numerous, and had an unusual arrangement; the physiological curves in this animal were equally anomalous. The change was permanently maintained.

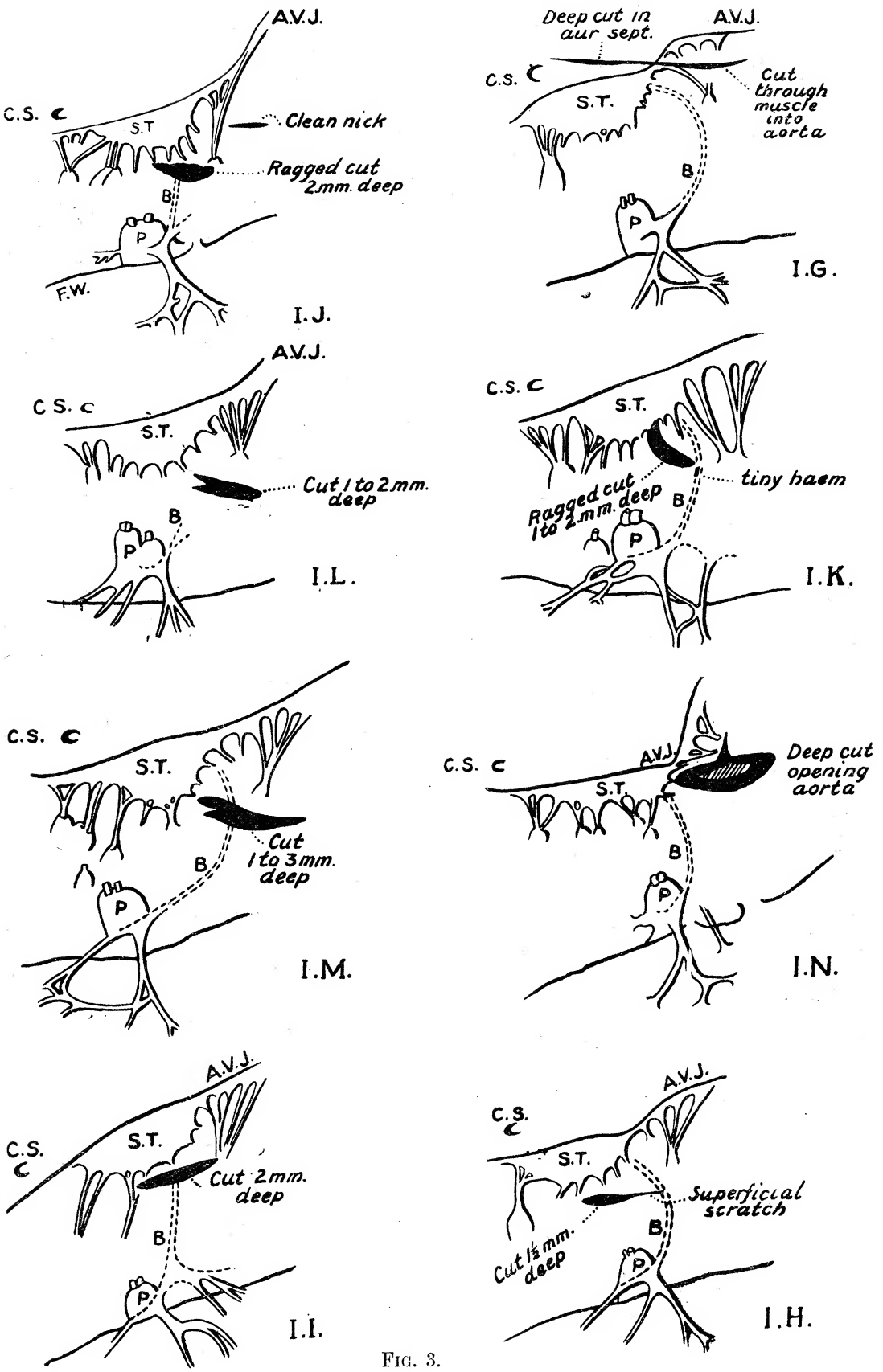

A.V.J.
C.S.
S.T
Clean nick
Ragged cut 2mm. deep
B
P
F.W.
I.J.
Deep cut in aur sept.
A.V.J.
C.S.
S.T.
Cut through muscle into aorta
B
P
I.G.
A.V.J.
C S.
S.T.
Cut 1 to 2mm. deep
B
P
I.L.
C.S.
S.T.
tiny haem
Ragged cut 1 to 2mm. deep
B
P
I.K.
C.S.
S.T.
Cut 1 to 3mm. deep
B
P
I.M.
C.S.
A.V.J.
S.T.
Deep cut opening aorta
B
P
I.N.
A.V.J.
C.S.
S.T.
Cut 2mm. deep
B
P
I.I.
A.V.J.
C.S.
S.T.
Superficial scratch
Cut 1½ mm. deep
B
P
I.H.

FIG. 3.

We pass to control observations.

Dog I.G. (8·0 kgrm.). A long cut was made at too high a level, and the sections showed the bundle and its branches to be untouched. It ran from a point above the coronary sinus (*C.S.*) ventrally, cutting the auricular septum deeply, crossing the *A–V* junction, and, penetrating the muscle of the ventricle over a length of 16 mm., opened the root of the aorta. A single curve was secured from Lead *II*; it had a natural outline. The heart dilated before the remaining curves could be photographed, but the curve of Lead *III* was seen to have remained unaltered.

Dog I.K. (11·1 kgrm.). A particularly instructive experiment. A cut was made in the customary situation. It was placed too obliquely. It had torn the edge of the valve and fell just short of the bundle division, as was clear macroscopically and microscopically. After a brief interval, during which the electrocardiogram remained unchanged, perfectly characteristic curves suddenly developed. They persisted for an hour, when the natural form of curve was again resumed. The course of the bundle division was marked opposite the end of the incision by a minute hæmorrhage, whose outlines were continuous with those of the bundle branch. The continuity of the bundle division was undisturbed, but at its ventral edge the lesion had distorted the bundle by crushing it a little and allowing blood to effuse around the fibres.

Dog I.N. (10·4 kgrm.). Cuts which fell together were made in the septum. The muscle was penetrated in the whole thickness immediately ventral to the septal valve segment. The course of the right bundle branch could be traced throughout; it was not involved. Curves taken from Leads *II* and *III* immediately afterwards were of normal outline, and differed but little from the preliminary curves. The curves of Lead *I* were not recorded, but it was seen that *S* had deepened somewhat; but by this time the right ventricle was rapidly dilating on account of the leak from the aorta.

Dog I.H. (5·3 kgrm.). A doubtful experiment. A small cut was made below the septal valve. As a muscular cut, it fell short of the bundle division, but was prolonged as a superficial scratch which penetrated the endocardium only. This scratch just crossed the branch, whose fibres were intact histologically. Characteristic curves were developed immediately. I did not continue the experiment, and the permanency or otherwise of the effect was not decided.

These observations seem to me sufficient to prove beyond reasonable doubt the relation between right bundle division lesions and the curves which are held to correspond to them in the dog.

The Curves Characterising a Lesion of the Right Division.

The electrocardiograms obtained when the right division of the *A–V* bundle is damaged are variable in form within certain limits. In the first place, I propose to describe in detail the commonest type (Plate 19, figs. 17 and 18), and, subsequently, to notice its variants. The broad features of the common type have been described by Eppinger and Rothberger.

In Lead *I* the auricular complex is followed by a ventricular complex of small amplitude, comprising preliminary and chief deflections. The preliminary deflections consist usually of a small downward, followed by a small upward, excursion; the former (Q') has a value of 0 to 0·2 millivolt, the latter (R') of 0·1 to 0·3 millivolt (Table I). These preliminary deflections are succeeded by the first chief deflection, a downward excursion (S') of from 0·2 to 1·3 millivolt; and the second chief deflection (T') a rounded and prominent summit.

TABLE I.—Amplitudes of Deflections in Bigram and Corresponding Levogram.

Dog.	Bigram.									Levogram.									Method.
	Lead *I.*			Lead *II.*			Lead *III.*			Lead *I.*			Lead *II.*			Lead *III.*			
	Q.	R.	S.	Q.	R.	S.	Q.	R.	S.	Q′.	R′.	S′.	Q′.	R′.	S′.	Q′.	R′.	S′.	
H.V.	0	13	1	0	19	6	1	10	8	1	26	0	0	14	23	0	5	40	Clamp.
H.W.	1	13	0	0	23	2	tr.	9*	4	2	14	0	0	5	27	0	4	35	Clamp.
I.H.	0	10*	0	0	19	5	0	12*	8	0	5	2	0	8	16	0	7	18	Incision.
I.K.	tr.	11	1	0	20	4	tr.	7	12	tr.	6	1	tr.	10	23	1	7	25	Incision.
H.M.	0	7	0	0·5	21	3	0·5	15	6	0	5	0	1	11	29	1	9	32	Clamp.
H.P.	1	8*	0	2	17	4	1	12*	6	1	1·5	8	2·5	7	33	1·5	7	21	Clamp.
H.S.	0	3	2	tr.	5*	3	0	3*	1	0	1	3	tr.	3	26	0	2	25	Clamp.
H.T.	1·5	6*	0	1·5	3	5	1·5	4	10	1	1	6	0·5	6	24	tr.	4	19	Clamp.
I.J.	0	8	0	0	17	3	0	10*	4	0	3	6	0	6	23	0	3	18	Incision.
I.L.	0	5	0	0	13	0	0	7	0	0	2	2	0	6	5	0	3	3	Incision.
I.M.	0	4	0	0	8	1	0	5	0	0	3	6	0	3	19	0	2	11	Incision.
I.O.	—	—	—	—	—	—	—	—	—	1	1	13	tr.	8	23	0	9	15	Incision.

The amplitudes in this Table are expressed in tenths of a millivolt. Summits marked with an asterisk were double or notched. The chest was open while all the curves were taken.

In Lead *II*, two preliminary deflections are the rule; a small downward movement (Q') of value 0 to 0·25 millivolt is usual, and a summit (R') usually sharp, and of value 0·3 to 0·8 millivolt,* is constant. Then follow the chief deflections: S', a steep and prominent downward movement whose value usually falls between 2·0 and 3·0 millivolts, and the course of which is often broken by a little notch; T', invariably upwards and rounded, and of considerable amplitude.

Lead *III* gives an electrocardiogram which is very similar to that of Lead *II*, but its dimensions are less. Q' varies between 0 and 0·15 millivolt, and is not often present; R' varies between 0·2 and 0·9 millivolt; S', usually between 1·1 and 2·5 millivolts; T' has the same direction and almost the same prominence as in Lead *II*.

The general features of these curves which deserve stress are as follows:—

(1) The first chief deflection (S') is in the same direction, *i.e.*, downward, in all curves, and is usually most prominent in Lead *II*, a little less so in Lead *III*, while in Lead *I* it is diminutive by comparison. (2) The second chief deflection (T') is always opposite in direction to the first chief deflection. (3) The initial deflections (Q', R', S') occupy a considerable time interval, having a duration in Lead *II* of from 0·0708 to 0·1083 sec.; they comprise approximately one-third of the whole ventricular complex, and have almost twice the duration of the initial phases in the bigrams

* Occasionally the rise in the curve starts from a point so far below the base line that it fails to reach the latter.

of the same animals (see Table II). (4) The curves in Leads *II* and *III* have an amplitude which is much greater than those of the normal curves in the same animal.

TABLE II.—Time Lengths of Bigram and corresponding Levogram in Lead *II*.

Dog.	Bigram.			Levogram.			Weight.
	Heart rate.	Length of initial phases.	Length of whole complex.	Heart rate.	Length of initial phases.	Length of whole complex.	
							kgrm.
H.V.	120	0·0518	0·2678	94	0·1083	0·3754	7·1
H.W.	186	0·0426	0·1960	200	0·0986	0·2460	12·4
I.H.	125	0·0451	0·2826	117	0·0744	0·3098	5·3
I.K.	160	0·0443	0·2297	169	0·0901	0·2480	11·1
H.P.	174	0·0616	0·2280	174	0·0884	0·2458	35·0
H.S.	171	0·0459	0·2214	170	0·0888	0·2360	9·4
H.T.	169	0·0474	0·2280	145	0·0912	0·2817	13·0
I.J.	145	0·0431	0·2238	146	0·0773	0·2659	5·5
I.L.	120	0·0453	0·2420	109	0·0708	0·2958	5·0
I.M.	132	0·0401	0·2568	148	0·0887	0·2720	11·3
I.O.	—	—	—	132	0·0891	0·1796	8·4
H.M.	171	0·0502	0·2066	176	0·0807	0·2768	9·4
Average...	—	0·0470	0·2348	—	0·0872	0·2694	—

Now while these curves are discovered as a rule, important variations may be noted in Leads *I* and *III*. The usual type I shall term "concordant," because the chief phases are similar in direction in all leads. The less usual is the "discordant" type, when the chief phases are opposite in direction in Leads *I* and *III* (see Plate 19, fig. 16). In these it is the curve of Lead *I* which diverges most, the first chief phase (R') is upward, and may have an amplitude of 1·4 or 2·6 millivolt; it is followed by the second chief phase (T'), which is a downward deflection. When this type of curve prevails in Lead *I*, the curve of Lead *III* is not a diminished but an exaggerated picture of Lead *II*, and the amplitude of S' may reach 4·0 millivolts in Lead *III*.

Thus the general features of these curves are not identical with those already enumerated for those of the concordant type, though the two types resemble each other in many respects; the second and third numbered statements apply to the concordant and discordant curves equally, the fourth statement applies to the discordant type in all leads.

As we shall see in the sequel, the discordant or rarer type is identical with curves taken from the human subject, and ascribed by many writers to lesions of the right bundle division in man; and the most baffling section of the present work has been the search for the cause of these variations. At first, when I was in possession of

EPPINGER and ROTHBERGER'S reports and of curves of my own of the concordant type, I believed that these alone occurred, and sought an explanation of their divergence from the "discordant" human curves. It seemed possible that the opening of the chest wall in the dog might materially affect them, but this was found not to be the case; the curves remain unchanged in their broad outlines when the chest wall is restored layer by layer and the lungs are fully expanded by extracting all air from the pleural sacs (see fig. 6). Next it seemed to me that the position of the heart might be responsible for the difference and, having ascertained the natural lie of the heart by studying published sections of the human chest and my own sections of the dog's chest, I carried out experiments in which the plane of my triangularly disposed contacts was altered on the restored chest wall, so that their positions relative to the heart might be as similar as possible in man and dog. In these experiments, also, I failed to find an explanation; but while they were in progress the first "discordant" curves were taken from a dog in which a straightforward experimental procedure had been employed. It then became evident that the reason lies in the heart itself, and the problem narrowed itself from an explanation of the different type in dog and man, to an explanation of two different types in the dog.

The variation in the type of electrocardiograms under discussion is unquestionably due to a variation in the original distribution of the excitation wave in the left ventricle. At one stage I expected that it might be due to differences in the relation of the lesion to the conducting system, so that in one instance the whole right stem suffered, while in another early outgoing branches escaped; but I failed to obtain any histological evidence for this view, and noticed further that in those hearts from which discordant curves had been taken the arrangement of the free Purkinje strands in the left ventricle was unusual; though numerous, they were much shorter than is customary and, lying closer to the wall, bridged the cavity to a less extent. But the most conclusive evidence was found in the natural bigrams, taken before the bundle division was damaged (Table I). In all instances where the discordant type of levogram was discovered, and where the first chief phase was upward (R') in Lead *I*, R in the bigram of the same lead was unusually prominent, amounting to as much as 1·3 millivolt (Dog H.V. and H.W.), whereas the average value of R in this lead is but 0·8 millivolt when the curves showing large values are included. Knowing that the bigram is of dual construction, and that the value of R in the bigram is partly dependent upon the value of R' in the levogram (a statement which evidently applies not only to Lead *II* where it has been fully demonstrated, but also to the remaining leads), we at once recognise why the discordant type of curve with its tall R' in Lead *I* is associated with an unusually tall summit R in the bigram of the same lead. But more important for our present purpose is the clear demonstration, which this association affords, that the discordant type is not the result of any variation in experimental procedure;

pointing, as it does clearly, to a pre-existing difference in the distribution of the excitation wave.

I may terminate this section by stating that in the dog concordant and discordant types are not sharply separated, but that intermediate types are encountered.

*The Meaning of Levograms and the Path of the Excitation Wave in the Left Ventricle.**

Discordant Curves.—For reasons which will be apparent subsequently, I propose to consider in the first instance those levograms which are of the discordant type.

In fig. 4 the initial phases of curves from Leads *I*, *II* and *III* have been plotted above each other. The method of constructing these charts has been explained in dealing with the toad's heart. In the dog I use as standard lead for orientation a direct lead from left ventricle to chest wall. The curves are all taken while respiration is suspended in the inspiratory position. I have plotted the curves for the first six hundredths of a second. Now these curves correspond to true levograms up to a point which is indeterminate, namely, to the last point where they represent spread in the left ventricle only. In writing of the dual construction of the bigram, it has been explained that, when the path to one ventricle is rendered functionless, the excitation wave flowing to the contralateral ventricle begins to spread from it to the homolateral ventricle after some 0·0350 to 0·0400 sec.

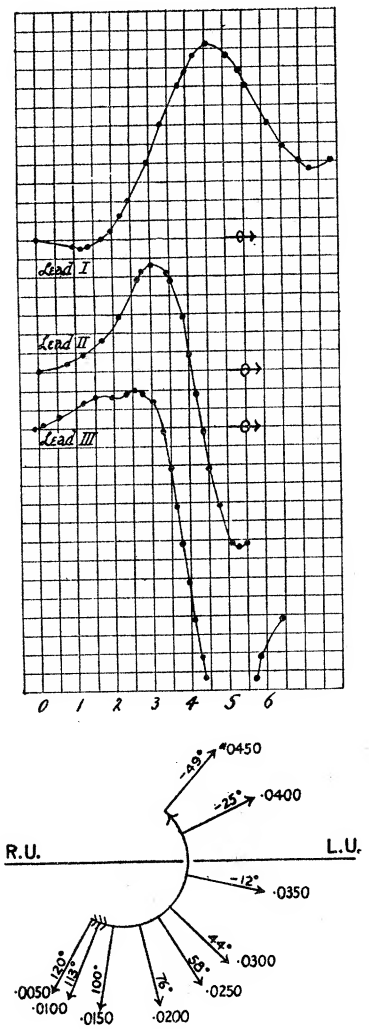


FIG. 4.—A chart showing the initial phases of the levogram, as it was recorded in Leads *I*, *II*, and *III* with the chest open (Dog H.V.). The curves are of the discordant type. The figures below the chart mark successive hundredth of a second lines. Ordinates, 1 cm. (four scale divisions) = 1 millivolt; abscissæ, 1 cm. (four scale divisions) = 0·02 sec.

Below is a diagram showing the electrical axes calculated for each 0·005 sec. The movement of the axis is uninterruptedly anticlockwise in its direction. The angles are expressed relative to the base line R.U.–L.U., which represents Lead *I*, from the right upper to the left upper limb of the animal. The data from which the angles were calculated are given in Table III.

or less subsequent to the inscription of the upstroke of *R*. The present charts are constructed so that the opening phase lies on the 0 line; consequently, the true

* A preliminary report of these observations appeared in the 'Proceedings of the Physiological Society,' May 15, 1915.

levogram is represented for some 0·0450 to 0·0500 sec. or less, the interval between the beginnings of the curves and *R* being approximately 0·010 sec.

The values of these three charted curves are given in the accompanying Table at each 0·0050 sec.; the curves fit, that is to say the values in Leads *I* and *III* are together equal to the values in Lead *II*. The corresponding angles, calculated trigonometrically, are recorded in the last column of the Table, and are drawn in the diagram beneath the chart (fig. 4). We see that the electrical axis is at first directed at an angle of 120° to the horizontal, and that it is rotated anticlockwise through 169°. The movement has not a uniform rapidity, being faster as the horizontal position is reached; but this is a minor detail—the chief fact is the uninterrupted character of the rotation; throughout it is in the stated direction. As in the case of the toad's heart, I judge this change in the direction of the electrical axis to express an alteration in the average direction in which the excitation wave moves. Interpreting the movement of the electrical axis in this wise, we may regard the diagram of rotation as expressing the change in the average direction of the excitation wave from instant to instant; and may read in the early directions the passage of the excitation wave downward and to the right in the septum; later its passage downward, or downward and to the left, at the apex of the ventricle; later its passage to the left in the lateral wall; and lastly its passage upwards and to the left in the lateral wall near the ventricular base.

TABLE III.—Dog H.V. (Levograms).

	I.	II.	III.	Angle.
sec.				
0·0050	−1	1	2·5 2·0	120°
0·0100	−2	3	5	113
0·0150	−1·5	6·5	8	100
0·0200	2·5	11 10·5	8	76
0·0250	11	21	10	58
0·0300	21	28	7	44
0·0350	35	24	−11	12
0·0400	44 45	4	−41	−25
0·0450	51	−25	−76*	−49

The potential values in this and succeeding Tables, under columns I, II, and III, are expressed in 0·05 of a millivolt.

* Calculated; Lead *III* off plate.

This interpretation agrees in a very precise fashion with the observations which ROTHSCHILD and myself have already published upon direct readings from the inner and outer surfaces of the ventricle, and with our general hypothesis that in the mammalian heart the excitation wave is distributed through the Purkinje system, and penetrates the ventricular musculature in a radiating fashion from the network. The curved arrow of the present diagram approximately represents the lie and

direction of the Purkinje paths in a coronal plane.* The direction of the axis at given time phases corresponds in a very satisfactory manner with the actual time readings of endocardial and epicardial distribution of the excitatory process. We may choose from the readings given by ROTHSCHILD and myself, time readings which express in a broad fashion the distribution in the left ventricle. The readings for the lining of the left ventricle are a little uncertain, but we considered it probable that the excitatory process arrives in the cephalic end of the septum some hundredth of a second or more before the upstroke of R; while it reaches the apex of the ventricle at or about the time when R starts. On the outer surface the readings are affected by the thickness of the muscle; but, in general, they increase from apex to base, and increase to an extent which can be accounted for only by the disposition of the conducting tracts. The surface excitation wave appears at the base of the left ventricle at about 0·0250 sec. after R. Representative readings are utilised in the accompanying diagram (fig. 5); and to harmonise these with all the time phases of

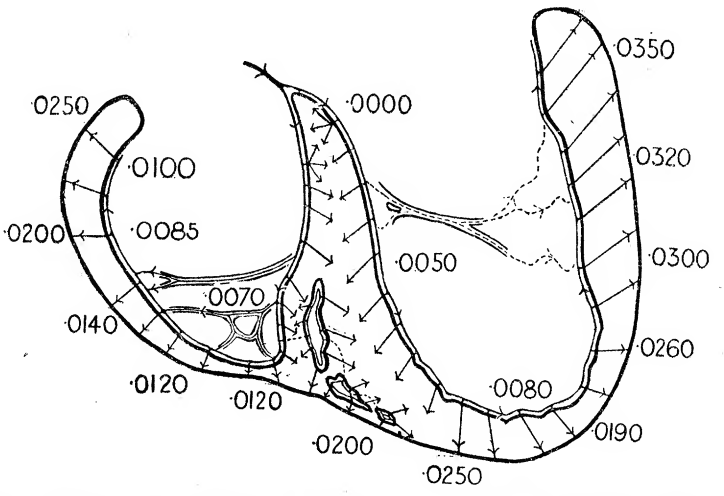


FIG. 5.—A natural size diagram of the dog's ventricle seen in section and illustrating the directions taken by the excitation wave in the spread through this heart chamber. The broken lines indicate spread through paths which do not lie in the actual plane of section. The figures illustrate direct readings in seconds from the epicardial and endocardial surfaces, and are related to the commencement of spread in the ventricle. The interval between this time instant and the beginning of R in the bigram may be taken at 0·0100 sec.

our chart (fig. 4) 0·0100 sec. has been added in each instance, being a fair allowance for the time interval between the beginning of R (to which the readings of ROTHSCHILD and myself were expressed) and the beginning of the levogram (which starts on the 0 line of our chart). My conception of the distribution would allow the excitation process to travel throughout the whole left ventricle in approximately 0·0350 sec.; commencing at 0 sec. in the cephalic end of the septum, it reaches the

* More correctly in a plane inclined to the coronal; the question of this rotation will be discussed a little later.

surface of the lateral wall at the ventricular base at 0·0350 sec., passing in an anti-clockwise fashion over septum to apex and later from apex to base over the free wall. Compare these readings with those of the movement of the axis in fig. 4 and a general agreement is observed, with the exception that the readings of fig. 4 are approximately 0·0100 sec. later than those of fig. 5 for corresponding time phases. This discrepancy is chiefly apparent near the end of the cycle, and is understood when we remember that the lining and surface readings represent the beginning of the rise of potential and not the full discharge. We read from the upstroke of the intrinsic deflection, not from its summit. The full discharge does not develop until at least 0·0100 sec. has elapsed from its commencement. There is, therefore, the nicest accord between surface readings and the direction of the electrical axis in the coronal plane; and this agreement is the strongest evidence of the correctness of my general hypothesis.

It becomes clear, if the hypothesis is sound, that the initial phases of the levogram in Lead *II** correspond to involvement of the septum in a left to right and base to apex direction, and that the summit R' corresponds to involvement of the apical musculature in a right to left and base to apex direction. The first chief phase (S'), on the other hand, represents involvement of the lateral wall, the direction in this structure altering gradually from a purely right to left to a right to left and apex to base direction.

Now R' of the levogram helps to build up R in the bigram, while S' is entirely responsible for S in the bigram.

Concordant Curves.—The movement of the electrical axis, corresponding to concordant curves, is not dissimilar to the movement just described. For the first 0·0250 sec. the movement in the two types is often almost identical. Thus in the example (fig. 6 and Table IV) the electrical axis starts at 150°, and moves in an uninterrupted anticlockwise fashion to −104°. In fig. 4 the movement up to 0·0200 sec. is to 76°; in the present instance it is to 77° (chest wall open). In fig. 4, at 0·0250 sec. the angle is 58°; in our present figure 30°. It is from this point onwards that the axis of concordant curves diverges from that of the discordant type. The axis moves rapidly across the horizontal 0° and takes up a position in the neighbourhood of −90°. In these concordant curves S' is almost equally prominent in Leads *II* and *III*, and it comes relatively early (at 0·0400 sec., as opposed to 0·500 sec. in fig. 4). The essential difference between the distribution in the concordant and discordant types is that in the former the large mass of muscle at the base of the left ventricle, the mass which is the last to be excited, is thrown into action earlier. I attribute this difference to the long free strands of Purkinje tissue which bridge the left ventricular cavity in these hearts (dotted paths in fig. 5). The bridging strands must inevitably affect the distribution of the excitation wave in the left ventricle, but their arrangement varies from heart to heart, and the

* Some levograms show the first spread to be horizontally to the right or even upward.

actual variations which they engender are in all instances difficult to picture. But the feature of levograms which it is most important for us to recognise is the invariable tendency to an anticlockwise rotation of the corresponding electrical axis.

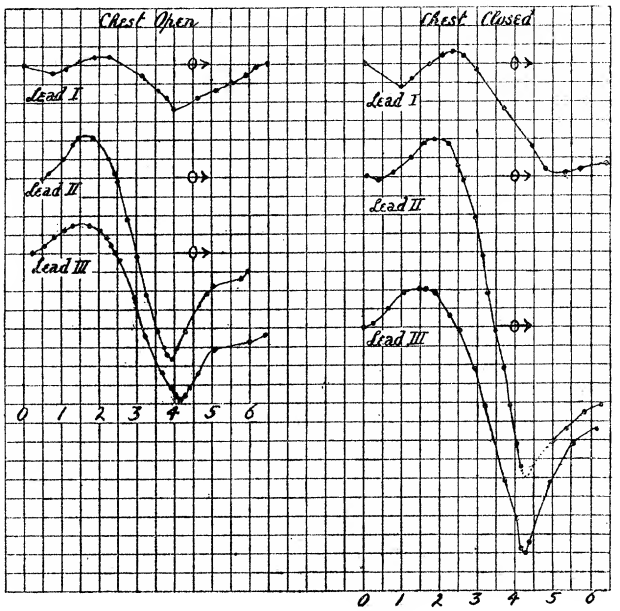


FIG. 6.—Charts showing the initial phases of the levogram in Dog H.T., and constructed in similar fashion to those of fig. 4. The curves are of the concordant type. The left-hand series was taken with the chest wall open; the right-hand series with the chest wall closed, and all air withdrawn from the pleural sacs through rubber tubes introduced for that purpose. In taking a single series of curves, the curve from each lead is taken simultaneously with a standard curve from a direct left ventricular lead. When the chest wall is to be closed, a long rubber tube filled with wool soaked in kaolin and passing through the chest wall, is employed as the ventricular contact. As is customary, the curves increase in amplitude, but are not materially altered in outline when the chest wall is closed. The angles formed by corresponding electrical axes are given in Table IV. Ordinates, 1 cm. (four scale divisions) = 1 millivolt; abscissæ, 1 cm. (four scale divisions) = 0·02 sec.

TABLE IV.—Dog H.T. Levograms.

	Chest open.				Chest closed.			
	I.	II.	III.	Angle.	I.	II.	III.	Angle.
sec.								
0·0050	−1·5	0	1·5	150°	−3	0	3	150°
0·0100	−1·5	4·5 4	5·5	103	−6	3	9	131
0·0150	1	11 9	8	84	−2	8	10	101
0·0200	2	9 8·5	6·5	77	2	10	8	79
0·0250	1	0	−1	?−30	3	3	0	30
0·0300	−2·5	−18·5 17·5	−15	−98	−2	−14	−12	−98
0·0350	−7	−38 36	−29	−101	−9	−41	−32	−102
0·0400	−12	−48 49	−37	−104	−16	−67 66	−50	−104

In both the cited instances this rotation has been uninterrupted; in some instances there is not quite the same degree of uniformity in the direction of movement; there may be for short time intervals a little to and fro movement about a given angle (fig. 8 and Tables V and VII). Eventually, however, the axis resumes its accustomed revolution.

TABLE V.—Dog H.P. Levograms.

	Rotated.				Coronal.			
	I.	II.	III.	Angle.	I.	II.	III.	Angle.
sec.								
0·0100	−2	−5	−3	−113°	−2	−5	−3	−113°
0·0150	1	7	6	82	0	4·5	4·5	90
0·0200	3	16	13	80	3	13	10	77
0·0250	0	12	12	90	2	11	9	80
0·0300	−3	0	3	? 150	0	0	0	
0·0350	−3	−11 10	−7	−107	−1	−16	−15	−94
0·0400	−2	−22	−20	−95	−5	−37 36	−31	−97
0·0450	−5	−37	−32	−97	−9	−43	−34	−101
0·0500	−12	−55 53	−41	−102	−15	−55	−40	−105
0·0550	−20	−64 60	−39 40	109	−22	−62	−40	−110

A final word completes this subject. I attempt to explain the rotation of the axis by assuming the passage of the excitation wave along the Purkinje paths of septum, apex and lateral wall in that order. As a matter of fact, however, the plane of our contacts has not the relation to the heart shown in fig. 5. Here the plane of section is at right angles to the septum; this plane in the dog makes with the coronal plane an angle of some 45–50°. In other words, the plane of the septum itself is not antero-posterior in the body, but is inclined to it, being approximately midway between the sagittal and coronal planes. Nevertheless, the electrocardiograms taken in the coronal plane clearly bear the impress of events in this mid-plane. If, while the chest wall is closed, we take two series of curves, the contacts being in the one instance in the coronal plane (Table V, coronal), and in the other instance being in a plane at right angles to the septum (Table V, rotated), the difference between the two series of curves is insignificant. It is clear therefore that the inclination of the plane of the septum does not vitiate the argument, or weigh materially against the general hypothesis. While some muscle elements excited along lines parallel to the plane of contacts will have, relatively, a preponderating influence upon the curves, while some excited in lines vertical to that plane will have, relatively, little influence, the main mass of muscle will be excited along lines of intermediate inclination; the electrical axis at a given instant will have a direction determined by complex factors, including the average direction of travel in all regions in process of activation and the masses of the muscular elements so involved. The

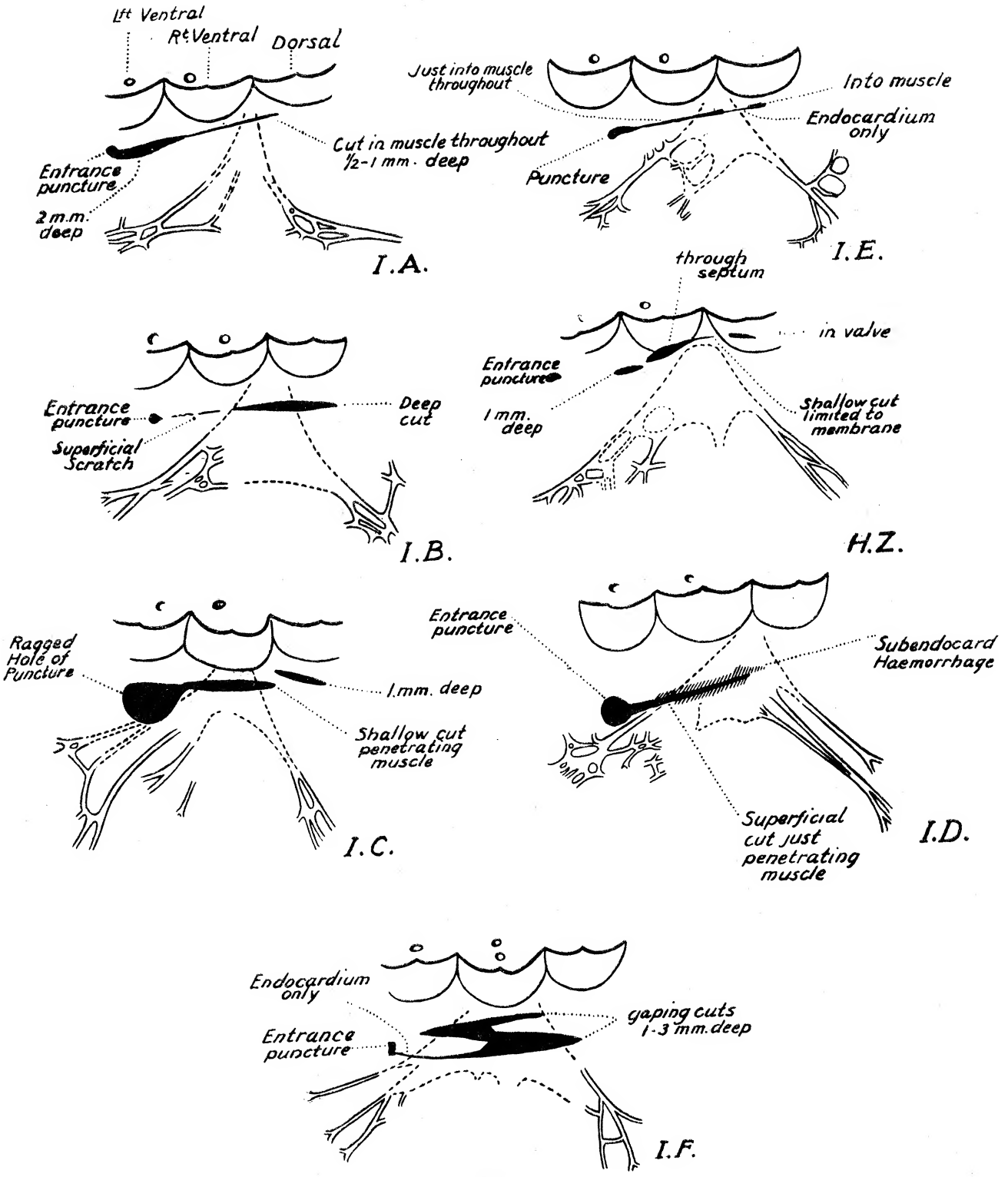

Lft Ventral
Rt Ventral
Dorsal
Entrance puncture
2 m.m. deep
Cut in muscle throughout ½-1 mm. deep
I.A.
Just into muscle throughout
Into muscle
Endocardium only
Puncture
I.E.
Entrance puncture
Superficial Scratch
Deep cut
I.B.
through septum
in valve
Entrance puncture
1 mm. deep
Shallow cut limited to membrane
H.Z.
Ragged Hole of Puncture
1 mm. deep
Shallow cut penetrating muscle
I.C.
Entrance puncture
Subendocard Haemorrhage
Superficial cut just penetrating muscle
I.D.
Endocardium only
Entrance puncture
gaping cuts 1-3 mm. deep
I.F.

FIG. 7.

relation of the septum to the coronal plane is such that it will tend to emphasise the fanwise spread of the excitation wave to those parts of the free lateral wall which fall in the coronal plane.

The Change in the Electrocardiogram Produced by Lesions of the Left Bundle Division in Dogs.

Proof of the Association of the Lesion with a Particular Type of Electrocardiogram.

As is well known, the left division of the A–V bundle in the dog passes over and lies on the muscular septum and, appearing in an almost constant position a little way beneath the junction of two aortic cusps—the right ventral cusp (from which the right coronary artery springs) and the dorsal cusp (from which no artery arises)—it occupies a purely sub-endocardial position, and quickly spreads and flattens itself out to form a fan (see fig. 7), the outlines and many details of which are clearly visible in the freshly opened heart. The borders of this fan are continued into its two most prominent subdivisions,* the ventral and dorsal branches, which run a free course from septum to the bases of the corresponding papillary muscle. Usually originating as single free strands, they are subject to minor variations. Near their terminations in the sub-endocardial spaces overlying the papillary muscles they break up into a complex basketwork. These chief facts are illustrated in the accompanying diagrams, where free strands encased in endocardium are indicated by unbroken lines, and where the remaining visible outlines of the fan are represented by broken lines.

In cutting the left division I use a small tenotomy knife, introducing it, as did Eppinger and Rothberger, from the ventral and left surface of the left ventricle. The point chosen is the crutch formed by the descending branch of the left coronary artery at its union with its chief branch, an artery which runs down toward the left and dorsal surfaces of the heart's apex. The knife is driven cleanly in between the two arteries, in a dorsal direction, with an inclination to the right; it enters the cavity $\frac{1}{2}$–1 cm. below the left anterior aortic cusp (see fig. 7), and in the space between the mitral valve and septum. The point is carried back until the dorsal border of the system is reached, when the cutting surface is opposed to the septum. A light pressure is exerted and is maintained, the knife being withdrawn *during the progress of the next systole* into the hole of entrance.

The figure illustrates seven experiments. In two preliminary experiments in which another route was adopted, several short and ugly lacerations of the septum, evidently failing to transect the bundle division, were without appreciable influence upon the electrocardiogram. The illustrations have been constructed from the freshly excised specimens and are accurately to scale. They may be described in the order in which they are figured. The description of the lesions is based upon the fresh appearance and subsequent histological examination in each instance.

* The remaining subdivisions course down the septum.

Dog I.A. (10·1 kgrm.). A single cut was made. The lesion included the entrance puncture, a wound 1 cm. in length and 2 mm. deep ventral to the bundle division, a shallow cut of 2 cm. extent and varying from $\frac{1}{2}$ to 1 mm. in depth, which ran upwards and included the whole of the fan at its base and transected it, as serial sections showed, completely. These wounds were all continuous in one line. The muscle was reached and cut along the whole line.

As a result the electrocardiogram immediately changed, the changes being perfectly characteristic in type, and being maintained permanently.

Dog I.B. (7·35 kgrm.). The lesion extended along a line of 3·4 cm. extent, and consisted of a small and isolated puncture, a superficial scratch ventral to the conducting strand and a deep clean incision of 1·8 cm. extent traversing the base of the fan completely. Macroscopically, the ventral extremity of the incision alone gave rise to doubt; the microscopic examination showed that the edge of the fan had been overlapped by the incision to a greater extent than is shown in the diagram.

Characteristic curves immediately developed and were maintained permanently.

Dog I.C. (15·8 kgrm.). The hole of puncture was long and ragged, involving the ventral border of the fan; running into it was a cut, appearing to penetrate the muscle along its whole length, crossing the base of the fan and dividing cleanly all but a few fibres at the dorsal margin; these showed evidence of having been crushed, and were densely infiltrated with extravasated blood corpuscles.

Characteristic curves (the initial phases of which are charted in fig. 9) immediately developed and were maintained.

Dog I.F. (8·1 kgrm.). Characteristic curves developed, and these persisted. The lesion was extensive, consisting of the hole of puncture and gaping cuts arranged in the fashion of a Z, which everywhere reached muscle. The bundle division was completely transected.

In the remaining experiments the incisions were placed less accurately, but these experiments are valuable as having supplied admirable controls.

Dog I.E. (7·6 kgrm.). The knife was inserted and withdrawn; the curves taken before and after the incision were of natural form, the two sets varying from each other in small details only. While the second set was being completed, characteristic curves of a left bundle lesion appeared. As soon as these were photographed, the natural form of electrocardiogram was resumed and persisted throughout the remainder of the experiment.

The lesion consisted of the hole of puncture continued into a shallow cut penetrating the muscle. It crossed the ventral border of the fan and cut the bundle division through two-thirds of its breadth. A short incision of similar depth was found in line with it, past the dorsal border of the fan, the two incisions being joined by an extremely shallow cut penetrating the endocardium only. The continuity of the bundle was intact over a considerable area.

Dog H.Z. (13·9 kgrm.). The broken cut was placed a little too high. In serial sections the cut was found immediately above the bundle bifurcation and the origin of the left division; its distance from the bundle varied from $\frac{1}{2}$ to 1 mm. in most places; at one point the incision came nearer to the division of the bundle, slicing off a very small piece of the tissue from the upper margin of the saddle. Some blood had effused into the tissues overlying the bundle division and the left branch, and at one point seemed to have exerted pressure on these structures. The continuity of the majority of the bundle fibres, and the complete continuity of the left bundle division was clearly established, although the lesion was extensive, the tissues of the septum being penetrated at some places completely, at others almost completely.

The curves taken before and after the incisions presented no material differences in form, being natural. Large excursions of the string seemingly characteristic of a left branch defect were seen but not recorded from Lead *II* for a few seconds immediately after the incision.

Dog I.D. (9·4 kgrm.). For a short while after the incision had been made the natural curve in Lead *II* was maintained; then there was an abrupt change to the characteristic curves of a left bundle lesion. These persisted sufficiently to permit of their being recorded, when a more natural type was once more resumed and persisted.

The cut crossed the ventral border of the fan and divided two-thirds of its breadth, leaving the dorsal border uninjured. An area of subendocardial hæmorrhage, the extent of which is indicated by the shaded area of the diagram, surrounded the incision and extended dorsally for 2 mm., almost but not quite completing the transection.

The observations recorded, including as they do a very fortunate series of controls, seem to me sufficient to prove beyond question the relation between lesions of the left bundle division and the curves which are held to correspond to them in the dog.

The Curves Characterising a Lesion of the Left Division.

The curves characterising lesions of the left division (Plate 20, fig. 20) are variable in certain respects.

In Lead *I* the auricular complex is followed by a ventricular complex which is, in its broad outline, diphasic. There are, as a rule, no preliminary phases in any of the three leads. The first phase is directed upward, the second is directed downward* and is blunt (T''); the first phase, which is the more prominent of the two, is often divided; its amplitude varies between 0·8 and 2·2 millivolts (Table VI).

In Lead *II* the curve is also broadly diphasic, the first phase (R') being almost invariably divided at its summit. It has an amplitude varying between 2·5 and 5·0 millivolts. The second phase (T'') is very prominent, and has an invariable downward direction.

In Lead *III* the curve is of the same form but smaller. The first phase, frequently divided at its summit, has an amplitude varying between 1·2 and 3·3 millivolts. T'' is a prominent downward deflection.

The general features of these curves which are to be emphasised are as follows :—

1. The first chief deflection (R') is in the same direction, *i.e.* upward, in all leads, and is always of greatest prominence in Lead *II;* it is usually less prominent in Lead *I* than Lead *III*, but the amplitude may be equal in these leads, or the relation may be reversed exceptionally.

2. The second chief deflection (T'') is always downward, that is to say, of opposite sign to the first phase, with isolated exceptions in Lead *I*.

3. The initial deflections (usually consisting of R' alone) are long, having in Lead *II* a duration of from 0·0824 to 0·1133 sec.; they comprise approximately one-third of the whole ventricular complex, and have almost twice the duration of the initial phases in the bigrams of the same animals (see Table VII).

4. With rare exceptions the curves in Leads *II* and *III* have amplitudes considerably surpassing those of the corresponding bigrams.

* Exceptionally, and when the first phase is diminutive, the second phase may have an upward direction.

Exceptionally, preliminary phases are also encountered; these may take the form of minute depressions or more rarely distinct depressions or distinct elevations in Leads *I* and *II*. In Table VI preliminary depressions have been expressed in the columns *Q'*. As in the case of the levograms, I attribute the more prominent variations to differences in the distribution of the Purkinje substance.

TABLE VI.—Amplitude of Deflections in Bigram and corresponding Dextrogram.

Dog.	Bigram.									Dextrogram.					
	Lead *I.*			Lead *II.*			Lead *III.*			Lead *I.*		Lead *II.*		Lead *III.*	
	Q.	R.	S.	Q.	R.	S.	Q.	R.	S.	Q'.	R'.	Q'.	R'.	Q'.	R'.
H.R.	0	9	0	0	35	0	2	16	0	0	8	0	25	0	24
I.A.	3	18	0	2	23	3	0	14	5	4	22	5	42	1	22
I.B.	tr.	4	1	0	29	3	0	27	3	0	18	0	50	0	33
I.C.	3	17	0	2	23	0	tr.	14	5	0	9	0	25	0	17
I.D.	1	20	0	0	23	5	0	13	9	1	22	0	45	0	28
I.E.	0	18	1	0	15	11	0	10	16	0	12	0*	37	0	33
I.F.	—	—	—	—	—	—	—	—	—	0	13	0	25	0	12

The amplitudes are expressed in tenths of a millivolt.

* In this animal a preliminary spike was shown in Lead *II*.

TABLE VII.—Time Lengths of Bigram and corresponding Dextrogram in Lead *II*.

Dog.	Bigram.			Dextrogram.			Weight.
	Heart rate.	Length of initial phases.	Length of whole complex.	Heart rate.	Length of initial phases.	Length of whole complex.	
							kgrm.
H.R.	186	0·0477	0·2046	190	0·0846	0·2018	16·2
I.A.	115	0·0538	0·2836	96	0·1011	0·2750	10·1
I.B.	125	0·0466	0·2846	83	0·1133	0·2970	7·3
I.C.	200	0·0444	0·1948	132	0·0824	0·2621	15·8
I.D.	114	0·0500	0·3080	120	0·0918	0·3152	9·4
I.E.	68	0·0590	0·3338	58	0·1030	0·3658	7·6
I.F.	—	—	—	136	0·0890	0·2874	8·1
Average.	—	0·0502	0·2682	—	0·0950	0·2864	—

The Meaning of the Dextrogram and the Path Taken by the Excitation Wave in the Right Ventricle.

The initial phases of the dextrogram have been plotted from a number of experiments. These curves represent true dextrograms up to a time phase lying some 0·0350 to 0·0400 sec. after *R*, that is to say, up to 0·0350 to 0·0400 sec. after the beginning of the *R'* in the dextrogram. Up to this point they may be utilised in calculating the direction of the natural electrical axis of the right ventricle. The common direction of the electrical axis of the right heart and its customary movements are represented most clearly in the first series plotted in fig. 9. Beginning at an angle of 30° there is a steady clockwise movement until the time 0·0250 sec. is reached; the angle of 70° or thereabouts is subsequently maintained. A clockwise movement at the first is the rule; the axis eventually sets between 50° and 90° in different experiments. Exceptionally the axis is almost steady between 70° and 90° from first to last; and in an isolated instance there was a slight anticlockwise movement.

The first direction of the axis is, as a rule, to the left and downwards; I interpret this to be the result of the response of the right side of the septum to impulses received from the right division of the bundle. Certain curves appear to indicate a preliminary upward movement in the septum, a phenomenon also witnessed at times in the left ventricle. The movement of the axis in a clockwise fashion I ascribe to gradual involvement of the lateral wall. The set of the axis to an angle approaching 70° seems to be accounted for by the general and simultaneous involvement of the free lateral wall and the trabeculated region. *A priori* it might be anticipated that a uniform clockwise movement of the axis would be found for the right ventricle, the excitatory process spreading down the septum and, later, gradually up the lateral wall. But this is not strictly the case. The architecture of right and left ventricles, and the distribution of the Purkinje arborisation to these two chambers, is different. We may exclude consideration of the conus, for in this structure the excitation wave progresses in planes almost at right angles to the plane of our contacts, and evidently can produce little effect upon the dextrogram; of the remainder of the right ventricular muscle it cannot be said that the basal muscle preponderates materially, as is the case in the left ventricle; for the apex of the right ventricle is blunt and consists of an extensive and heavy spongework, the trabeculated region. Our surface readings show us that the base of the left ventricle (in such a plane as is diagrammatised in fig. 5) receives the excitation wave little if at all earlier than the extreme apex of the right ventricle; when the axis sets it appears to express the component of the electrical forces which are set free by the almost simultaneous activities of these two muscle masses. The general direction in which the trabeculated region is activated, as judged by anatomical considerations, is strongly downwards and to the left; the general direction at the right base,

as judged anatomically and experimentally, is to the right and somewhat upward. The resultant electrical axis is downward. The general activation of the right ventricle in the dog in a downward direction results from the complete and extensive bridging of the cavity by free strands of Purkinje tissue (see fig. 5). Taking account simply of the free wall of the right ventricle, the uppermost free strands join this wall at or a little below its centre point; but if we consider the whole of the right ventricle, including the trabeculated region, which is also supplied by the right bundle division, then the uppermost free Purkinje strand approaches the base of the ventricle much more closely than the apex. A general movement of the excitation wave in a base-apex direction is therefore to be anticipated in the dog. The clockwise movement which obtains in the heart of man (to be described) is masked or destroyed in the dog by what may be spoken of as a short-circuiting of the apical regions of this ventricle, brought about by the conspicuous and numerous bridges traversing the right ventricular cavity in this animal.

In the axial dextrogram the curve *R* rises at first gradually, and subsequently with increased steepness. The first part of the rise is attributable to septal activity, later the rise is enhanced when the trabeculated region and the lower part of the free wall becomes involved. Selected readings from the paper by ROTHSCHILD and myself accord with the view here expressed (fig. 5). Adjusting our readings* to the present charts and diagrams, the first surface reading comes at about 0·0120 sec., the earliest full discharge on the surface† comes certainly as late as 0·0220 sec.

At this time phase, when the electrical axis is beginning to set, the surface discharge is developing fully in the "central region," namely, the region where the free wall is attached to septum. From this time onwards, till the full development of discharge at the base (0·0350–0·0400 sec.) and at the extreme apex (0·0350–0·0400 sec. or a little less), the direction of the axis remains almost constant.

THE INTERPRETATION OF THE INITIAL PHASES OF THE DOG'S BIGRAM.

We are now in a position to complete our survey of the initial phases of the natural electrocardiogram. The chief conclusions have already been foreshadowed. The natural electrocardiogram is of dual construction, consisting of a summation of levogram and dextrogram. This has been shown for Lead *II*, and its demonstration for the remaining leads is, strictly speaking, unnecessary. But if further proof of the conjoint character of the natural curve were desired, it would be found in such charts as are published in figs. 8 and 9. In these charts the initial phases of the bigram have also been plotted, the time phases of bigram and levogram (fig. 9) or of bigram

* Allowing 0·0100 sec. for the interval between the commencement of the excitation wave in the ventricle and the beginning of *R*.

† Judged by the distance from the beginning to the summit of the intrinsic deflection.

and dextrogram (fig. 9) corresponding accurately.* Possessing two of the three sets of curves, we may calculate the third; thus, if bigram and dextrogram are plotted from observation, the levogram can be ascertained algebraically (fig. 9). *The calculated levogram* (fig. 9) *and the calculated dextrogram* (fig. 8) *possess their*

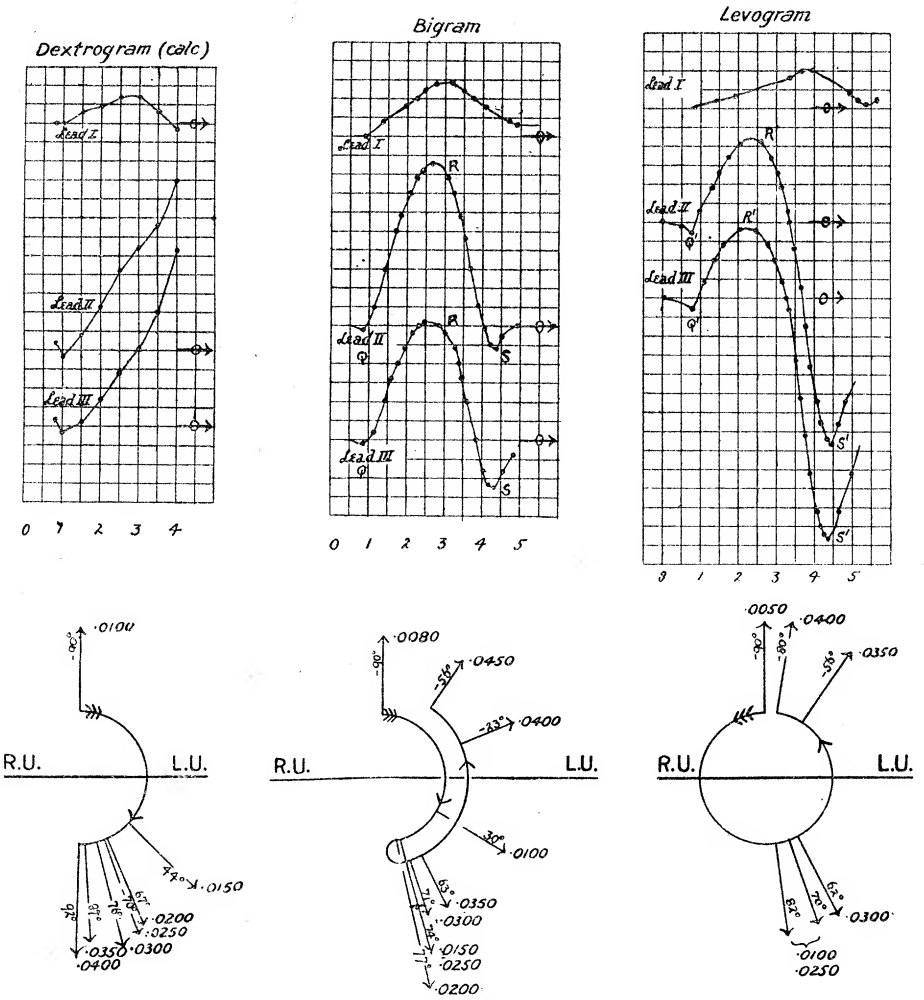


FIG. 8.—Three series of curves with corresponding diagrams of the electrical axes and their rotations (Dog H.M.). The left-hand series is of the calculated dextrograms, the central series of the actual bigrams, the right-hand series is of the actual levograms. The angles formed by the electrical axes are given in Table VIII. Ordinates, 1 cm. = 1 millivolt; abscissæ, 1 cm. = 0·02 sec.

usual outlines and magnitudes, and the directions of the electrical axes and their movements correspond closely with examples actually observed.

These complete charts are particularly serviceable in that they demonstrate the composition of the bigram in the clearest fashion. In fig. 8 the axis corresponding to the bigram moves at first in a clockwise, and later in an anticlockwise, fashion.

* The plotting against time is accomplished by maintaining a standard lead from the left ventricle while bigram and levogram are photographed, or from the right ventricle while bigram and dextrogram are photographed.

TABLE VIII.—Dog H.M.

	Levogram.				Bigram.				Calculated dextrogram.			
	I.	II.	III.	Angle.	I.	II.	III.	Angle.	I.	II.	III.	Angle.
sec.												
0·0050	0	−1	−1	−90°								
0·0080	—	—	—	—	0	−1	−1	−90°				
0·0100	1	3	2	70	1	1	0	?30	0	−2	−2	−90°
0·0150	2	13 14	12	82	5	18	13	74	3	4	1	44
0·0200	3·5	21 21·5	18	81	8	33	25	77	4·5	11·5	7	67
0·0250	5	21 22	17	77	12	42 43	31	74	7	21	14	70
0·0300	7	14 15	8	62	14	41 42	28	71	7	27	20	76
0·0350	9	−7	−16	−56	12	26	14	63	3	33	30	87
0·0400	9·5	−44	−53·5	−80	8	1	−7	−23	−1·5	45	46·5	92
0·0450	7	−57	−61 64	−84	5	−4	−9	−56	−2	53	55	88

The first, or clockwise movement, which happens while the upstroke of R is inscribed, is clearly governed by the dextrogram. The turning point, where clockwise gives place to anticlockwise movement, corresponds to the summit of R; it is at this instant that S' in the levogram begins, and from this point onwards the levogram governs the bigram, and the anticlockwise movement is maintained. In the second example (fig. 9 and Table IX) the bigram opens with an angle of −110°, evidently governed by the levogram; for the next 0·0300 sec. it is governed chiefly by the dextrogram; at its termination the levogram once more predominates.

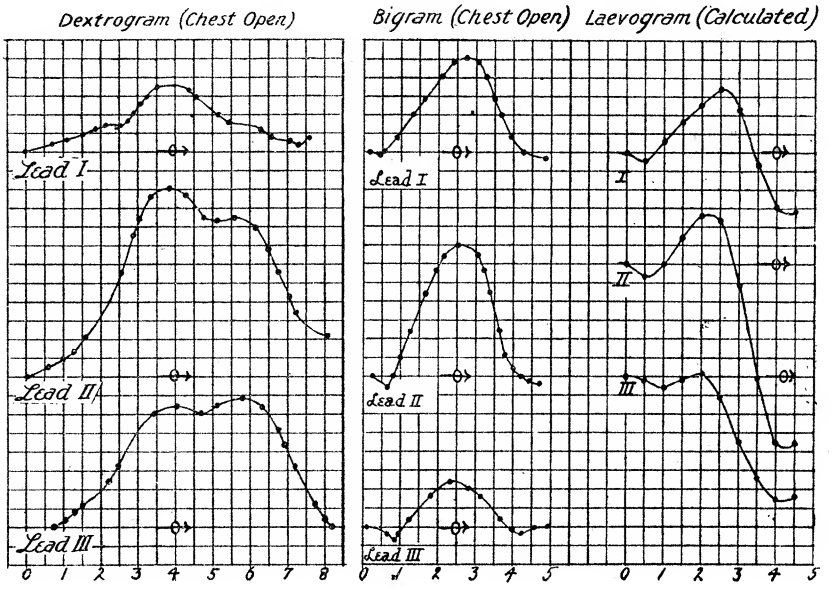


FIG. 9.—Four series of curves from Dog I.C. The left-hand series shows the initial phases of the actual dextrogram with chest open. The second series is that of the actual bigram. The third series is that of the levogram, calculated from the curves of dextrogram and bigram. The data from which the electrical axes were calculated are given in Table IX. Ordinates, 1 cm. = 1 millivolt; abscissæ, 1 cm. = 0·02 sec.

Table IX.—Dog I.C.

	Dextrogram. Chest open.				Dextrogram. Chest closed.				Bigram. Chest open.				Levogram (calculated). Chest open.			
	I.	II.	III.	Angle.	I.	II.	III.	Angle.	I.	II.	III.	Angle.	I.	II.	III.	Angle.
sec. 0·0050	2	2	0	30°	1	2·5	1·5	67°	−0·5	−1·5	−1	−110°	−2·5	−3·5	−1	−134°
0·0100	3	5	2	53	0	3 4	4	90	6	5	−1	21	3	0	−3	−30
0·0150	4	10	6	66	−1	9	10	95	12	18 17	5	47	8	7	−1	23
0·0200	6·5	16	9·5	66	1	18	17	87	19	29	10	50	12·5	13	0·5	32
0·0250	7	26 24·5	17·5	74	11	32·5 32	21	70	24	35 36	12	49	17	11·5	−5·5	12
0·0300	12·5	40 39	26·5	71	23	51	28	63	24	33	9	45	11·5	−6	−17·5	−50
0·0350	17·5	49 48·5	31	69	27	72	45	68	14	18	4	42	−3·5	−30·5	−27	−96
0·0400	18	50	32	69	28	83	55	71	3	2	−1	10	−15	−48	−33	−108
0·0450	15	46	31	71	27	82	55	71	−1	−2	−1	−120	−16	−48	−32	−109

In dealing with the toad and tortoise heart I have shown that the initial phases (*R* and may be *S*) cover that period of the heart's cycle during which the muscle mass, as a whole, is passing into the state of excitation. In the case of the dog's heart, I come to a similar conclusion; and it is based primarily upon the correspondence between the periods of the initial phases of levogram and dextrogram, respectively, and the periods covering readings from the surface and lining of the corresponding ventricles. But this statement is true only if we consider those phases which are parts of the true levogram and true dextrogram; that is to say, if the downstroke of *R'* in the dextrogram and a good deal of its plateau, and if the upstroke of *S'* in the levogram are not included. Treating the time-relations in a broad way, it is more satisfactory to deal with the bigram itself. The initial phases of this curve, stretching from the beginning of *Q* to the return of *S* to the base line, have an average duration of some 0·0480 sec. (see Tables II and VII). The readings of intrinsic deflections lie between 0·0000 and 0·0350 sec.; they show us that the whole ventricular muscle is activated within 0·0350 sec.; but the full discharge is not developed at the latest points to be activated until some 0·0100 sec. has elapsed. Thus the stage of complete activation, as estimated from direct readings, is approximately 0·0450 sec., a figure in close agreement with the length of the initial phases of the axial lead. *Thus the length of the initial phases of the bigram may be taken to indicate the duration of the process of activation.*

The initial phases of the levogram and of the dextrogram are of longer duration, and this is evidently due to the abnormality and late spread in right or left ventricle. When the right or left bundle division is cut, we may judge from the lengths of the initial phases before and after section, that the process of activation is retarded by some 0·0400 sec. (see Tables II and VII). For the initial phases of the levogram or dextrogram exceed those of corresponding bigrams by some 0·0400 sec. In strict accord with this estimate is the alteration in the values of readings over the right ventricle when the right bundle division is divided; they rise by approximately 0·0400 sec. (see figs. 12 and 13 of the paper by Rothschild and myself, and fig. 1 of the present monograph). There is a similar rise in the value of readings over the left ventricle, when the left bundle division is divided. Where does this delay occur? The main delay is not due to an altered direction of spread in the affected ventricle, though this factor is almost certainly in some small measure responsible; but it is conditioned by slow conduction across the septum, the path which the excitation wave must necessarily take in passing from one ventricle to the other when a bundle division is divided. This is evidenced not only by the almost uniform delay of the readings over the surface of the affected chamber, but by our direct observation of slow conduction across the septum (see previous communication). The plateau and fall of *R'* in the dextrogram and the slow rise of *S'* in the levogram are simultaneous with the abnormal and delayed spread in the left and right ventricle respectively. The close correspondence between the time period covering

a complete series of direct readings and the time period occupied by the initial phases, in a variety of animals and in hearts excited along different paths, contributes the weightiest evidence that our direct readings are reliable.

Finally, I may sum up* the origin of the initial deflections of the dog's electrocardiogram in conclusions which apply to all leads :—

1. *Q, the first deflection of many electrocardiograms, is due in most dogs to activation of the septum from the left bundle division;* the chief evidences for this statement are :—(*a*) that the earliest reading of an intrinsic deflection has been obtained from the septum beneath the aortic valves; (*b*) that the corresponding electrical axis has a usual direction from left to right† ; (*c*) that *Q* is almost always constituted by the levogram‡ ; (*d*) that *Q* is absent in axial electrocardiograms from the toad and tortoise, in which animals no septum exists.

To this origin of *Q* I attribute its relatively frequent appearance in Lead *I* in the dog.

2. *R is in greater part a right, and in lesser part a left, ventricular effect and is mainly due to the progress of the excitation wave down the septum and its adjoining structures.* This conclusion is based :—(*a*) upon the interpretation of *R′* in the levogram and dextrogram respectively : in the levogram *R′* represents an average movement of the excitation wave downward to the right, in the dextrogram a movement downward and to the left ; (*b*) upon the observation that the upstroke of *R* is written before any surface reading is obtained, is at its height when the surface potential is fully developed over the thinnest region of the right ventricle in the central region of the heart ; and (*c*) upon its consistency with such septal readings as have been obtained.

3. A division of *R*, or notching of *R*, is explained by lack of synchronism between the summit of *R′* in the levogram, and the second or main turning point ; the latter is due to a balance established between the ascending limb of *R′* in the dextrogram and the descending limb of *S′* in the levogram. These conclusions are based upon an analysis of the composite parts of bigrams.

4. *S is known to result from an upward movement of the excitation wave through the basal and lateral wall of the left ventricle,* because :—(*a*) *S* is always contributed solely by the levogram ; (*b*) it corresponds to an outwardly and upwardly directed electrical axis ; (*c*) the conclusion is consistent with surface readings from this part of the ventricle.

* A preliminary summary of this part of the monograph was published in the 'Proceedings of the Physiological Society,' July 3, 1915.

† In some instances the direction is almost vertically upwards ; the exact manner of *Q*'s production in these circumstances is somewhat uncertain.

‡ In some few instances the dextrogram apparently contributes in small measure.

APPENDIX.

The following illustrative experiment, while forming no essential part of the foregoing observations, has many interesting and indirect relations with them.

The ventral surface of a dog's heart being exposed (fig. 10), the muscle was excited at a number of points (1–14), using at each point successive threshold stimuli. The corresponding responses of the heart were recorded in Lead *II*, and single ventricular cycles are shown to the left in the figure. The signal of stimulation was also recorded (see Curve 13), and in each instance the time interval between the signal of stimulation and the first prominent deflection was measured. These intervals are tabulated (Table X). Subsequently the heart was hardened in a natural condition of diastole, and an oblique section was cut passing through the points of stimulation along the line 3–13. A diagram of this section is also represented in the same figure. The distance of the ventricular surface to the Purkinje network was measured in millimetres at all stimulated points.

TABLE X.

Point stimulated.	Distance to Purkinje system.	Signal to chief phase.
	mm.	sec.
1	R. 4	0·0363
2	R. 3	0·0416
3	R. 3	0·0424
4	R. 4	0·0405
5	R. 3	0·0407
6	R. 3·5	0·0473
7	R. 1·5	0·0293
8	R. 2	0·0245
9	R. 2	0·0343
10	R. 3	0·0497
11	R. 7 L. 8	0·0686
12	R. 12 L. 10	0·0920
13	L. 9·5	0·0920
14	L. 8	0·0699

If the series of curves is examined, it will be noticed that the ventricular responses obtained over the whole ventral surface of the right ventricle yield very similar outlines in the electrocardiogram; these vary from each other in detail and magnitude.* As the point of stimulation is moved along the A–V groove from the right towards the left margin and on to the conus, the excursion of the string, in its response to the heart beat, increases somewhat in magnitude. As the stimulating

* The curves are complicated by the presence of auricular complexes; these are of the type accompanying natural auricular beats in the case of Curves 3, 4, and 5; the remaining curves show auricular complexes of the retrograde type.

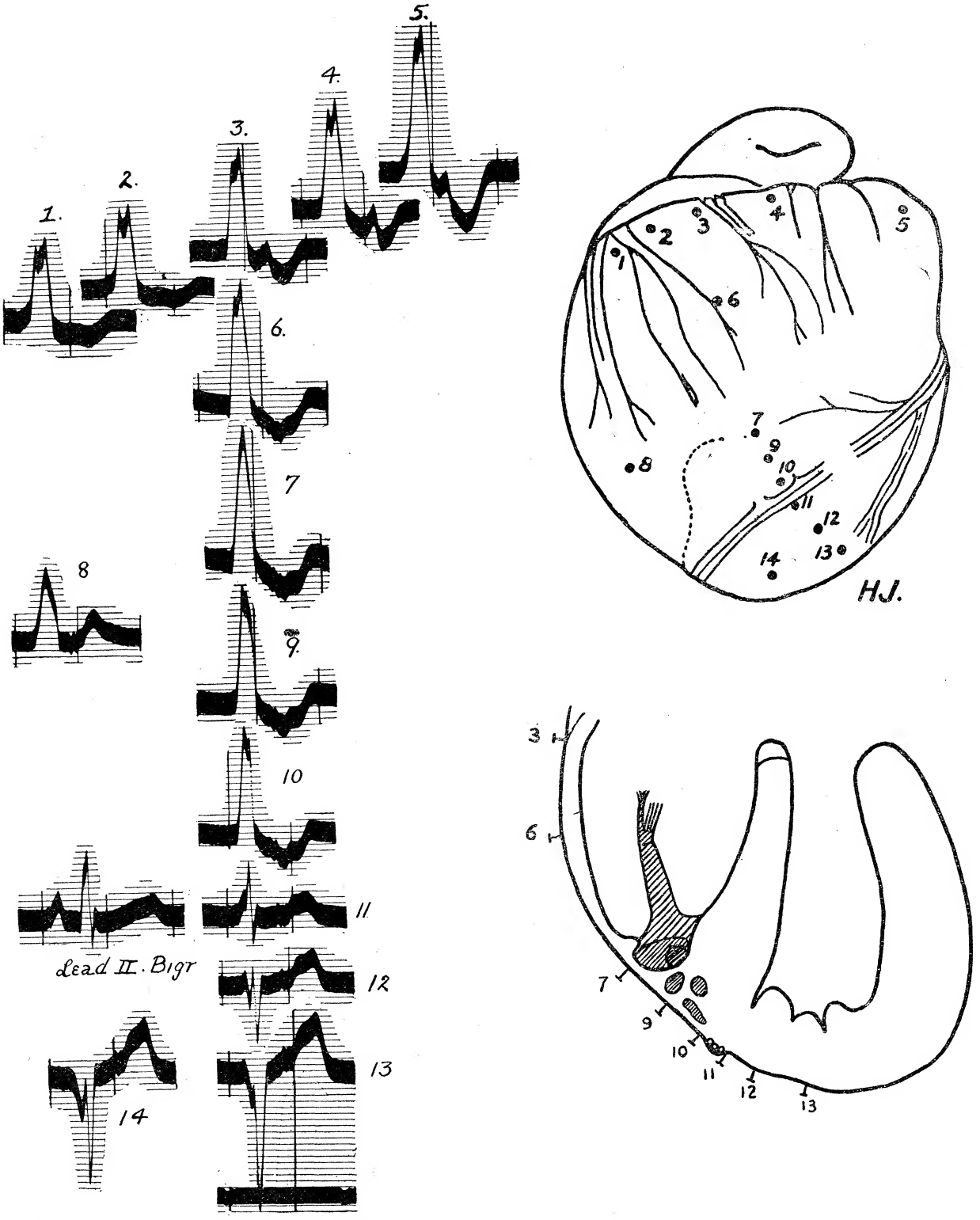


FIG. 10.—(Dog H.J.) A series of curves taken from Lead *II*, and resulting from stimuli applied at the corresponding numbered points in the upper outline drawing of the dog's heart. This outline is to scale and natural size. The lower outline is a section of the same heart, through points 3–13, and is also to scale.

electrodes are moved from the A–V groove (at 3) downwards towards the apex of the heart, the change in form and in magnitude is very slight until the trabeculated region is passed. As the descending branch of the left coronary artery is crossed there is an abrupt change in type; point 11 yields a complex of intermediate type; the complexes from points 12 and 13 have their two chief phases inverted as compared to those obtaining over the right heart. Briefly, the responses from the right ventricle produce electrocardiograms presenting the chief features of *dextrograms*, while the responses from the left ventricle produce electrocardiograms showing the chief features of *levograms*. Now these facts are not new in their entirety; the type of response has been extensively studied by many writers; I may refer to my own summary of such observations,* and to a recent paper by ROTHBERGER and WINTERBERG;† but several points of importance have hitherto escaped notice or explanation.

The first point to which I would call attention is the relation between the length of the interval, signal to first chief phase, and the thickness of the underlying muscle (Table X). In Curves 12 and 13, and preceding the chief or downwardly directed movement, there are very evident preliminary phases of a diphasic character. In Curves 4, 7, 9 and 10 the complex opens with a steep upstroke, and there is no preliminary phase, but small preliminary phases are distinct in all the remaining curves.‡

ROTHSCHILD and I have shown that when the surface of the ventricle is stimulated, the excitation wave quickly spreads to the Purkinje network and reaching it is propagated at an accelerated velocity. The preliminary phases of the present curves are due to the initial spread in the muscle: for, being more or less prominent according as the muscle path is long or short, we may conclude that one of the chief factors governing their appearance is the amount of muscle activated before the Purkinje network begins to convey the impulse. The first chief phase, be it upward or downward, appears immediately after the Purkinje system is involved, and its magnitude is due to quick spread to the relatively large and corresponding muscle area. In this fashion I interpret the relation between the muscle thickness and the interval, signal to chief phase.

The manner of spread when the surface of the ventricle is stimulated is diagrammatised in fig. 11.

The excitation wave spreads from the point of stimulation in all directions, and the muscle mass involved is approximately hemispherical. This hemisphere expands until the whole thickness of the wall is involved; up to this moment the string, at the usual tensions, remains quiescent or shows only minute movements. It

* 'Mechanism of the Heart Beat,' London, 1911, p. 146.

† 'Archiv. f. d. ges. Physiol.,' vol. 154, p. 571 (1913).

‡ The preliminary phases are included within the tabulated intervals, the ends of these intervals falling where the preliminary phases terminate.

is clear that a wide area of muscle wall may be thrown into a state of activity before the recording instrument is affected; this fact requires emphasis because it has to be taken into account in interpreting the natural axial electrocardiogram.

The next phase of the spread is more complex, the excitation wave having entered

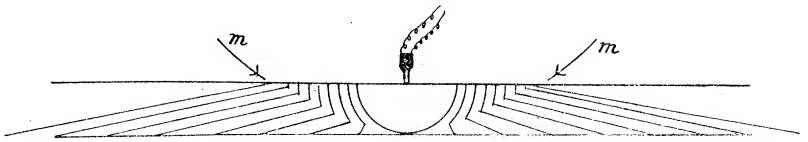


FIG. 11.—A figure illustrating the supposed lines of spread of the excitation wave when a point upon the surface of the ventricle is stimulated.

the Purkinje substance courses rapidly along the lining of the heart and re-enters the muscle from within outwards. From this time onwards the excitation wave is spreading along two routes: along the old path, which is radial to the point of stimulation, and along the new path from the network. If we consider a point in the region *m*, such a point may be activated by the surface wave, or it may be activated by the wave travelling from within outwards. All points on the surface within the region *mm* will be activated directly from the point of stimulation; all points outside this region will be activated from the network. A little consideration will suffice to show that, when the surface of a ventricle is stimulated, only a limited mass of muscle responds to the ingoing wave of excitation; the chief part of the ventricular wall responds to the outgoing wave. The mass responding to the ingoing wave will depend upon the muscle thickness, but will never be large, because the velocity of conduction in Purkinje tissue is relatively very great. Thus it happens that the direction of travel in the ventricular tissue cannot be controlled by altering the point of stimulation. Stimulation at base or apex of the right ventricle produces the same end result, an excitation wave travelling from within outwards over the greater part of the ventricular substance. This accounts for the general similarity of curves obtained when stimulating a large area of the right, or when stimulating a large area of the left ventricular surface.*

The second point to emphasise is the relatively abrupt transition from the type shown in Curve 10 to that shown in Curve 12. Clearly such transitions are due to spread into the right Purkinje system on the one hand and into the left Purkinje system on the other.

Finally, when the point stimulated is immediately to the left of the artery (point 11) curves of intermediate type are obtained, and these curves often resemble natural bigrams in the same animal very closely, as ROTHBERGER and WINTERBERG and others have pointed out. The explanation is that the two Purkinje systems are involved almost simultaneously.† To take the present example,

* ROTHBERGER and WINTERBERG have suspected a mechanism of the kind described, and briefly discuss its possibility in their article.

† A parallel though less exact explanation was given by the writers cited

the resemblance between Curve 11 and that which stands to the left of it in the figure (*i.e.* the natural bigram) is remarkable. On measurement the point of stimulation was calculated to lie 7 mm. from the nearest Purkinje strands in the right ventricle, and 8 mm. from the nearest strand in the left ventricle. The electrocardiogram, shown in Curve 11, is of dual origin; it is an algebraic summation of a dextrogram and levogram, and has the general outline of the natural bigram. That it fails to reproduce the natural bigram with absolute accuracy is not surprising, for the dextrogram and the levogram which compose it are not quite natural, neither is it probable that they fall with precisely natural time relations to each other. Nevertheless, the experiment corroborates the observations recorded in an earlier section of this communication.

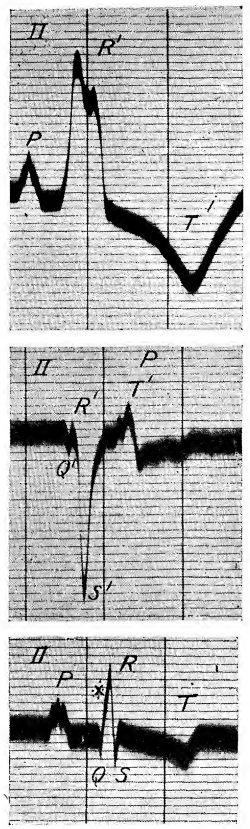


FIG. 12.

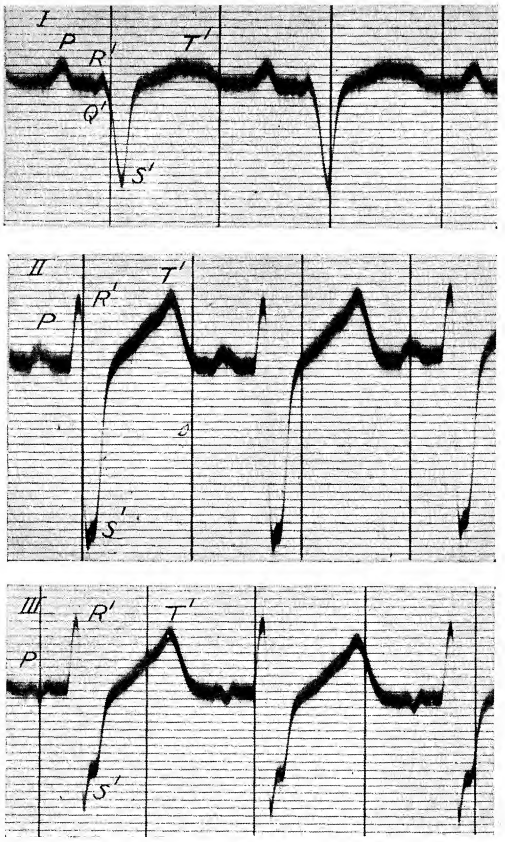


FIG. 13.

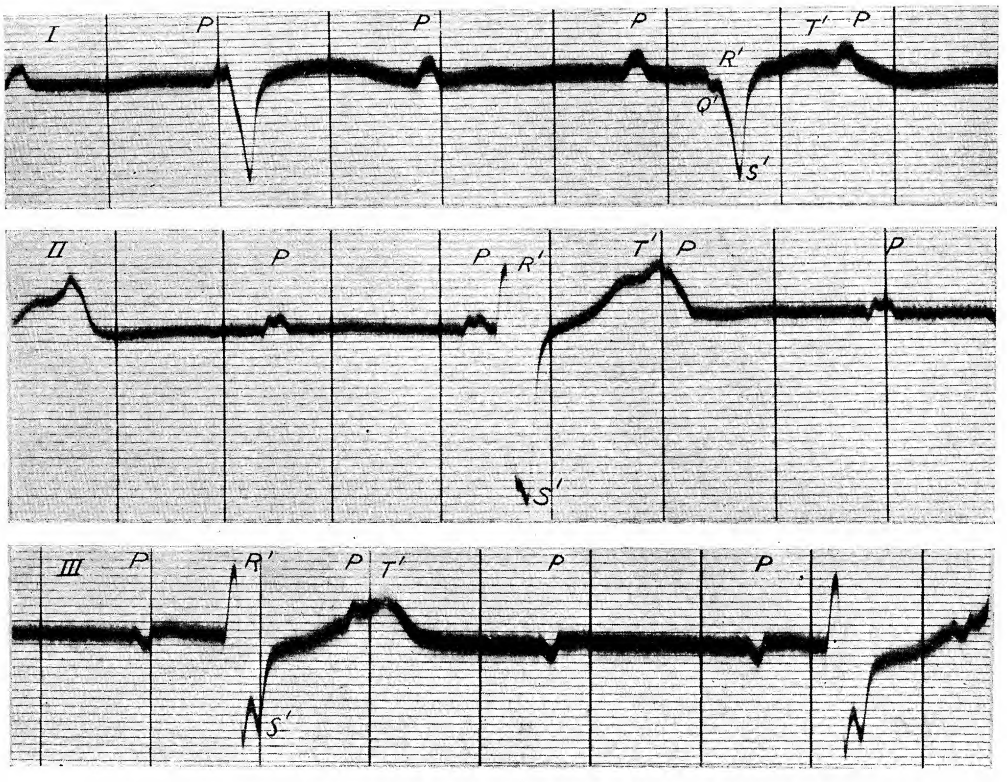


FIG. 14.

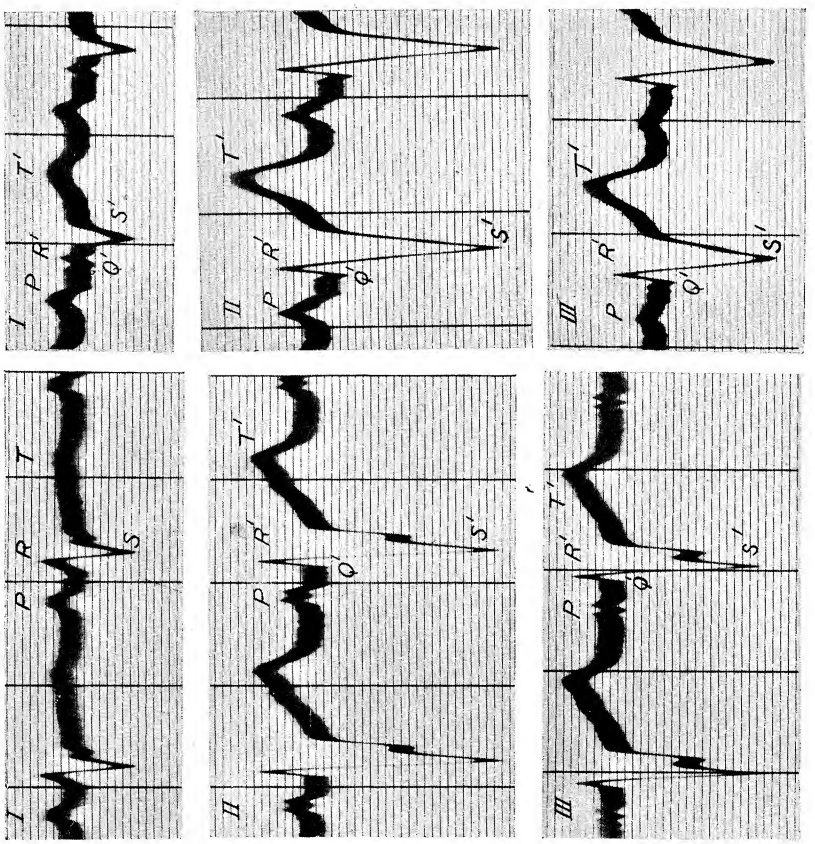


FIG. 18.

FIG. 17.

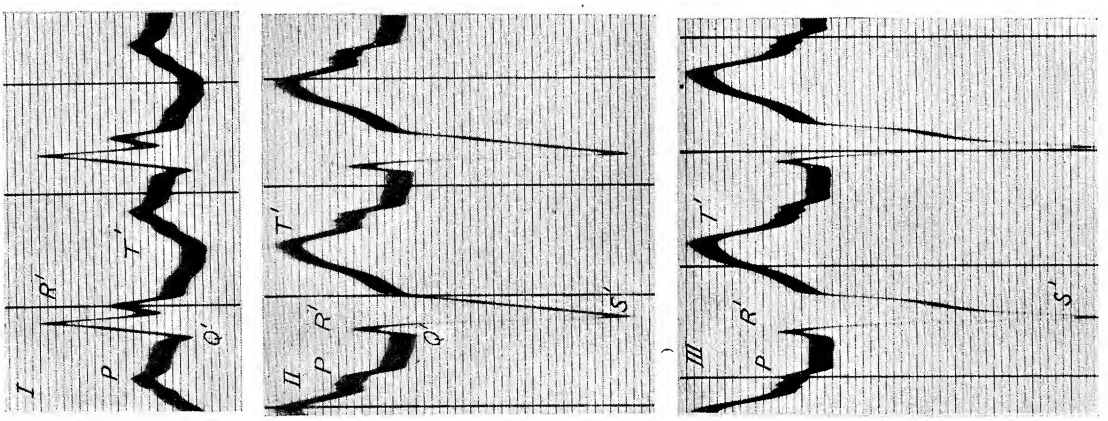


FIG. 16.

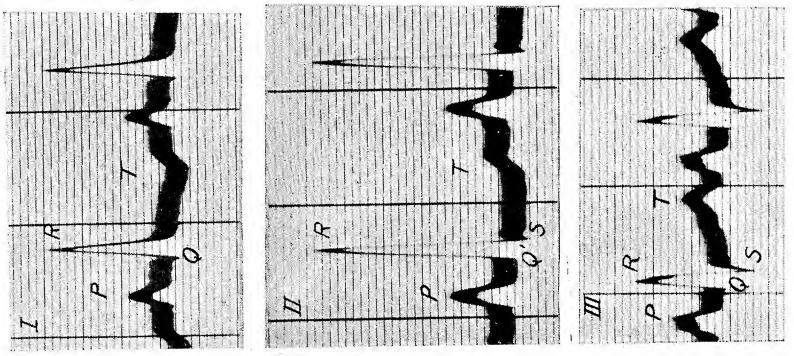


FIG. 15.

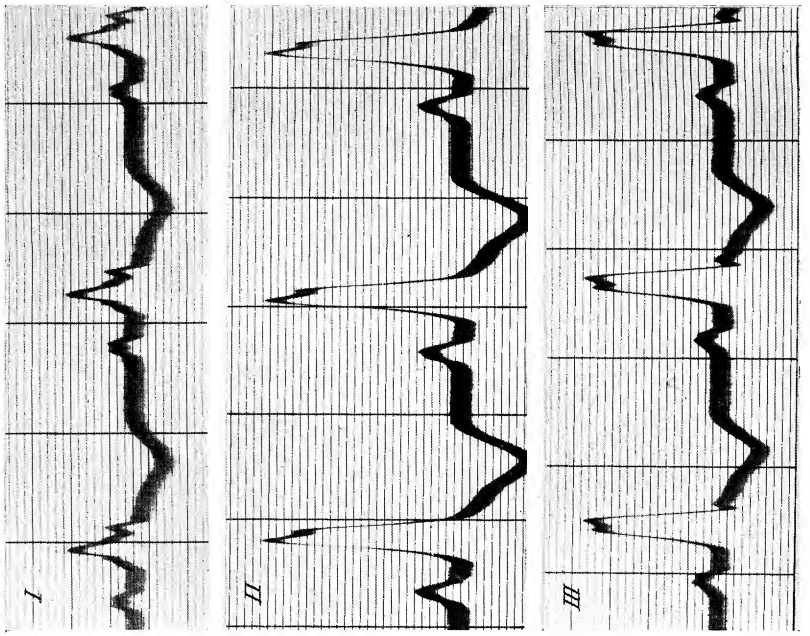


FIG. 20.

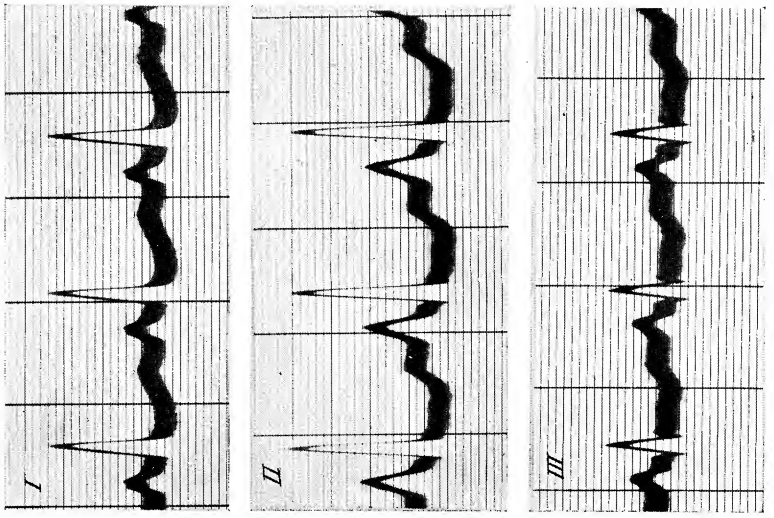


FIG. 19.

EXPLANATION OF PLATES.

Plate 18.

Fig. 12.—(Dog H.H.) Three electrocardiograms from Lead *II*. Top curve dextrogram, middle curve levogram, bottom curve bigram. While the dextrogram and levogram were recorded, complete heart-block was present. These three curves were used in the construction of fig. 2 (p. 251). Ordinates, approximately, 1 cm. = 1 millivolt; abscissæ = 0·2 sec.

Fig. 13.—Levograms from Leads *I*, *II*, and *III* (Dog I.O.), the result of transection of the right bundle division. Ordinates, approximately, 1 cm. = 1 millivolt; abscissæ = 0·2 sec.

Fig. 14.—The corresponding levograms, after clamping the *A–V* bundle and producing complete heart-block. The outlines of the ventricular curves in this and the preceding figure are essentially the same. Ordinates, approximately, 1 cm. = 1 millivolt; abscissæ = 0·2 sec.

Plate 19.

Fig. 15.—(Dog H.W.). Electrocardiograms from Leads *I*, *II* and *III*, chest wall open. Showing the curves of the natural heart beat. Ordinates, approximately, 1 cm. = 1 millivolt; abscissæ = 0·2 sec.

Fig. 16.—(Dog H.W.). Electrocardiograms from Leads *I*, *II* and *III*, chest wall open. Showing the form of the levogram, obtained by clamping the right bundle division in the same animal. The curves are of the discordant type. Ordinates, approximately, 1 cm. = 1 millivolt; abscissæ = 0·2 sec.

Fig. 17.—(Dog I.J.) Electrocardiograms from Leads *I*, *II* and *III*, chest wall open; levograms of concordant type produced by transection of right bundle division. Ordinates, approximately, 1 cm. = 1 millivolt; abscissæ = 0·2 sec.

Fig. 18.—(Dog H.P.) Electrocardiograms from Leads *I*, *II* and *III*, chest wall open; levograms of concordant type produced by clamping right bundle division. Ordinates, approximately, 2 cm. = 3 millivolts; abscissæ = 0·2 sec.

Plate 20.

Fig. 19.—(Dog I.C.) Electrocardiograms from Leads *I*, *II* and *III*, chest wall open. Showing the curves of the natural heart beat. Ordinates, approximately, 1 cm. = 1 millivolt; abscissæ = 0·2 sec.

Fig. 20.—(Dog I.C.) Electrocardiograms from the same animal and from Leads *I*, *II* and *III*, showing the form of the dextrogram, produced by transection of the left bundle division. Chest wall open. Ordinates, approximately, 1 cm. = 1 millivolt; abscissæ = 0·2 sec.

Part IV.—*The Human Ventricle.*

[PLATE 21.]

Aberrant Contractions.

Shortly after EPPINGER had published his experiments with ROTHBERGER, the same writer recorded some clinical observations in conjunction with STOERK.* In electrocardiograms taken from Leads *I* and *III*, in two patients, they observed abnormalities of the curves which, so they considered, represented defective conduction in the right division of the *A–V* bundle; they obtained *post-mortem* examinations in these two cases, and observed fibrosis of the ventricular septum, which involved the right bundle division in each.

The anomalous curves first depicted by EPPINGER and STOERK are very familiar to those who are accustomed to deal with many cases of heart disease. I have in my collection over thirty examples. Three years ago I described those features of the curves which I regarded as characteristic,† and accepting EPPINGER'S explanation I termed the ventricular contractions corresponding to them "aberrant contractions."

CARTER has since published from my laboratory a detailed account of the patients on whom I then based my conclusions,‡ and has included in his paper the curves of 17 of our cases.

In the monograph to which reference has been made, I said that "the chief features of the ventricular complexes are the exaggerated amplitude of excursion, and the prolongation of the initial phases. The deflections which replace the usual *QRS* group have a total duration which exceeds a tenth of a second, and generally comprise more than a third of the whole complex. When the *right* branch of the bundle is at fault, the abnormality declares itself in the broad summit (R') of Lead *I* and in the deep and broad depression (S') in Lead *III*. Damage of the *left* branch portrays itself in the reversed pictures, a deep broad depression (S') in Lead *I*, and a tall and broad summit (R') in Lead *III*." I also pointed out that, while the chief phase is opposite in direction in Leads *I* and *III*, the curve in each lead is in the main diphasic, so that the second phase (T') is invariably in the opposite direction to the chief initial phase. In Lead *II* the curves are not so characteristic; here they are smaller in amplitude than in Lead *III*, but usually follow the general outline of that lead. There is little to add at the present time to this description, with the exception that the chief phase in the damage of the right stem is usually preceded in Leads *II* and *III* by a small or insignificant upward phase, and in Lead *I* by a small downward phase. The curves must be studied in

* 'Zeitschr. f. klin. Med.,' vol. 71, p. 157 (1910).

† 'Clinical Electrocardiography,' London, 1913, p. 32.

‡ 'Archives of Internal Medicine,' vol. 13, p. 803 (1914).

detail and in numbers if the significance of my present observations is fully to be appreciated.

For my immediate purpose I take two examples, one series of curves which I regard as a characteristic example of a right branch defect in the human subject, the other the solitary example of curves in my possession which I regard as representing a left branch defect (Plate 21, figs. 11 and 14). These two sets of curves, taken during a clinical routine, are unaccompanied by standard simultaneous curves; and to place similar phases of time above each other is not quite so simple as would have been the case had such simultaneous curves been in my possession. The initial

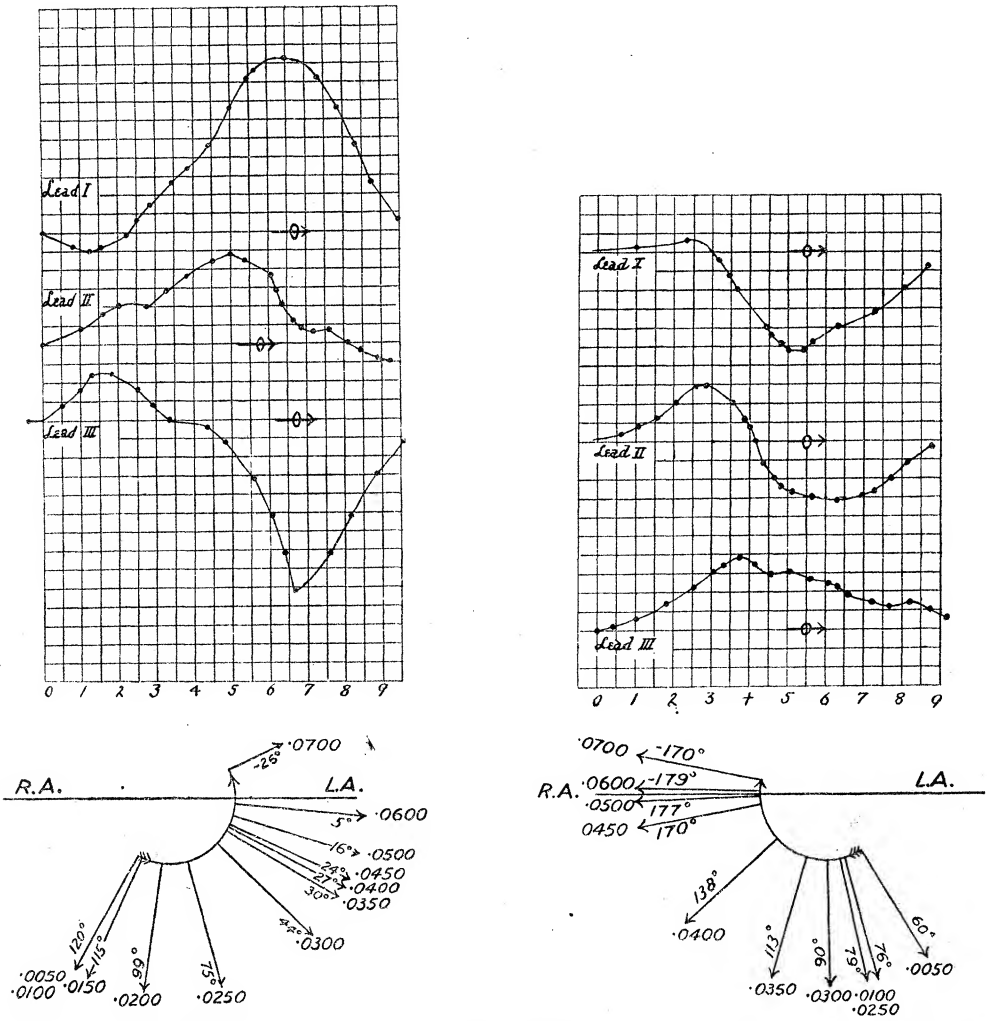


FIG. 1.—A chart of the initial phases of human electrocardiograms, taken from a heart presenting signs of a right bundle branch lesion. Ordinates, 1 cm. = 1 millivolt; abscissæ, 1 cm. = 0·02 sec. A diagram showing the angles of the corresponding electrical axes and the rotation. The actual curves are shown in Plate 21, fig. 11; the values are given in Table I.

FIG. 2.—A similar chart and diagram, taken from a heart presenting signs of a left bundle branch lesion. Ordinates and abscissæ as in fig. 1. The actual curves are shown in Plate 21, fig. 14; the values are given in Table II.

phases of the three leads have been charted in the case of each series, and have been moved relative to each other until a fit, in the sense of an agreement with EINTHOVEN'S formula, has been obtained. This, though an arduous task, is no less certain than the other method, for agreement with the formula can be obtained only when corresponding time phases have been found. The charts are illustrated in figs. 1 and 2, and beneath each chart is a corresponding diagram of the changing direction of the electrical axis. The rotation in the case of the supposed right branch defect (fig. 1) is a very complete and tolerably uniform anticlockwise movement; the rotation in the case of the supposed left branch defect (fig. 2) is a very complete and almost uniform clockwise movement.

TABLE I.—Human Levogram.

	I.	II.	III.	Angle.
sec.				
0	0	0	0	—
0·0050	−2	2	4	120°
0·0100	−4	4	8	120
0·0150	−5	7	12	115
0·0200	−2	10	11 12	99
0·0250	3	10 11	8	75
0·0300	9	11 12	3	44
0·0350	14	15 14	0	30
0·0400	19	19 18	−1	27
0·0450	24 25	22	−3	24
0·0500	33 32	24	−8	16
0·0600	45 44	19	−25	5
0·0700	45 46	3	−43	−26
0·0800	30	1	−29	−28
0·0900	9	−5	−13 14	−50

In this and succeeding Tables the figures representing voltages in the several leads express 0·05 millivolt.

TABLE II.—Human Dextrogram.

	I.	II.	III.	Angle.
sec.				
0·0050	1	2	1	60°
0·0100	1	4	3	76
0·0150	1·5	6·5	5	77
0·0200	2	10	8	79
0·0250	3	14	11	78
0·0300	0	14	15 14	90
0·0350	−7	10·5	18 17·5	113
0·0400	−14	4	18	138
0·0450	−22 23	−8	15	170
0·0500	−26 28	−13	15·5 15·0	177
0·0600	−22 25	−15 13	12	−179
0·0700	−17 20	−15 13	7	−170

The charted and figured curves with the diagrams of rotated axes may be compared with those of fig. 3 and the angles of Table III, taken from a large Rhesus monkey, in which a lesion of the right bundle branch had been created experimentally.* The comparison leaves little or no doubt that these human curves and those from the monkey have a similar constitution, and convinces us that we may utilise the human curves as a basis in analysing natural human electrocardiograms. At the present time we have an abundance of those curves which represent right

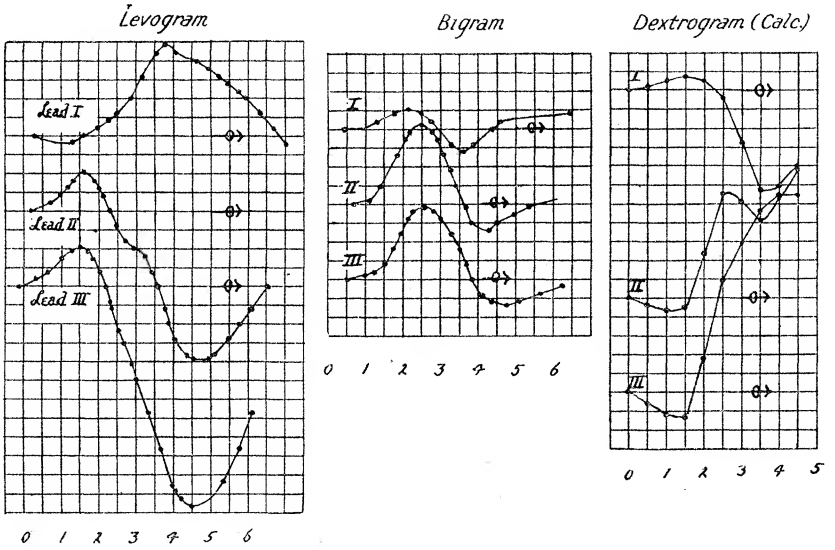


FIG. 3.—A chart showing three sets of curves obtained from a large Rhesus monkey. The series to the left is that obtained after clamping the right division of the bundle. The central series comprises the natural curves obtained from the undamaged heart. The right-hand series shows the calculated dextrograms. Abscissæ and ordinates as in fig. 1. The values of the curves are given in Table III.

TABLE III.—Monkey.

	Levogram.				Bigram.				Dextrogram (calculated).			
	I.	II.	III.	Angle.	I.	II.	III.	Angle.	I.	II.	III.	Angle.
sec.												
0·0050	−1	2	3	109°	0	0	0	—	1	−2	−3	−71°
0·0100	−2	5	7·5 7·0	106	0·5	1 1·5	1	71°	2·5	−3·5	−6	−65
0·0150	−0·5	10	10·5	92	3	6·5 7	4	65	3·5	−3	−6·5	−57
0·0200	2	6	4	71	4·5	17 17·5	13	76	2·5	11·5	9	78
0·0225	4	1	−3	−16								
0·0250	6	−5	−11	−57	4	22 22·5	18·5	80	−2	27·5	29·5	93
0·0300	13	−10·5	−24 23·5	−56	−1	15	16	93	−14	25·5	39·5	110
0·0350	21	−17·5	−38·5	−57	−5·5	3	8 8·5	130	−26·5	20·5	47	124
0·0400	22·5	−32·5	−54 55	−65	−3	−6	−3	−120	−25·5	26·5	52	119
0·0450	20·5	−39	−58 59·5	−70	1	−5	−6	−81	−19·5	34	53·5	111

* The lesion was confirmed histologically.

branch defects; we have an insufficiency of those which represent left branch defects. Certain conclusions can be recorded nevertheless.

The human levogram—of which we are evidently now entitled to speak with precision—usually begins in Lead *I* in a minute downstroke. *Q* appearing in the human bigram in this lead probably represents spread in the left ventricle. In Leads *II* and *III* the beginning is with a small upstroke; *Q* in the human bigram in Leads *II* and *III* probably represents spread in the right ventricle. In the dog, on the other hand, we have seen it to be a rule that *Q* when it appears in the bigram almost always expresses the commencing activity of the left ventricle.

We may best judge of the time-interval taken for the excitatory wave to complete its circuit in the human heart, from the length of the initial phases of the bigram. The intervals for the separate ventricles are similar, amounting to 0·05–0·07 second. The aberrant curve is a true representation of levogram or dextrogram only so far as the full excursion of its chief phase; that is to say, in the levogram, to the top of *R′* in Lead *I* and to the bottom of *S′* in Lead *III*. The prolongation of the initial phases as a whole is due, as in the dog, to abnormal subsequent spread in the opposite ventricle.

The conclusions in respect of notching of *R* in the dog's electrocardiogram and to other variations in form may be applied equally to the human electrocardiogram.

It will be evident that Rothberger and Winterberg's conclusion that none of these anomalous curves can be regarded as expressing a right or left branch defect* unless the chief phases are in the same direction in Leads *I* and *III* is not sound. It is based upon their findings in the dog. As we have seen, the chief phases of Leads *I* and *III* usually obey this rule in this animal; but even in the dog there is a good deal of latitude in respect of form, and the two chief phases may be opposite in sign. Rothberger and Winterberg have fallen into more serious error in extending their conclusion to the human subject; for in man, as in the monkey, the chief phases in Leads *I* and *III* are of opposite sign.

In the human curve, the passage of the excitation wave through the left ventricle is mainly responsible for *R* in Lead *I* and *S* in Lead *III*; while its passage through the right ventricle is chiefly responsible for *S* in Lead *I* and *R* in Lead *III*. The importance of this evident conclusion will be emphasised immediately in our study of the curves of hypertrophy in the human heart.

Curves of Hypertrophy.

Since Einthoven first drew attention to certain abnormal types of electrocardiogram, and with the accumulation of fresh evidences, opinion has gradually hardened to the belief that relative preponderance of the right or left ventricle may be recognised in human electrocardiograms. Recently, the evidences for this view

* 'Zentralb. f. Herzkrank.,' vol. 5, p. 206 (1913).

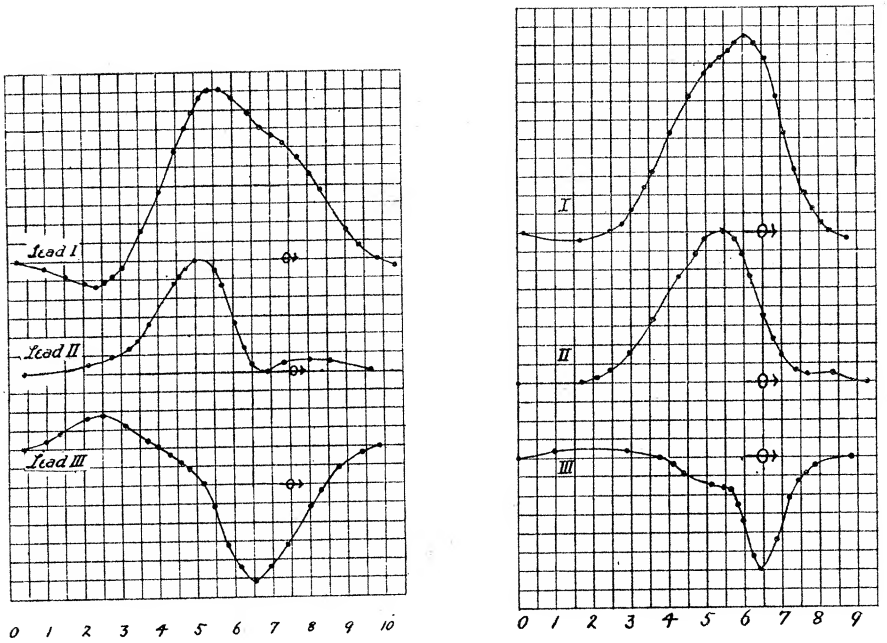
have been summed up and amplified.* Relative preponderance of the left ventricle is represented by an increase in the amplitude of *R* in Lead *I* and of *S* in Lead *III*; while relative preponderance of the right ventricle is represented by an increase in the amplitude of *S* in Lead *I* and *R* in Lead *III*. These are the prominent signs. It is not with this fact but with its explanation that we are concerned at present.

It would seem clear that if a single ventricle becomes enlarged in all its dimensions, the general distribution of the excitation wave throughout it will remain unaltered. But as the mass of tissue involved in the process of excitation is increased, the electric forces liberated will suffer corresponding exaggeration. There appears to be no reason why hypertrophy of the left ventricle should materially modify the general shape of the levogram, but there is reason to anticipate that such a change will magnify the amplitudes of the several deflections. It is to be expected, *a priori*, that the curves from subjects who present preponderance of the left ventricle will exhibit a preponderance of the levogram; the levogram, as we have seen, is mainly responsible for *R* in Lead *I* and for *S* in Lead *III*. An exaggerated *R* in Lead *I*, an exaggerated *S* in Lead *III*, are to be predicted—they are actually discovered—in instances of left ventricular preponderance. Similarly, an exaggerated *S* in Lead *I*, an exaggerated *R* in Lead *III*, are to be predicted—and are actually found—in instances of right ventricular preponderance.† Hypothesis is, to this extent, substantiated by observation. It is further substantiated by more detailed inquiry.

If the curves of left hypertrophy are curves in which there is a notable predominance of the levogram, the rotation of the electrical axis in curves of left hypertrophy should be similar to the rotation of the axis corresponding to the levogram. That is to say, it should be anticlockwise. I have charted the electrocardiograms from a case of chronic renal disease and from two instances of severe aortic disease (figs. 4 and 5), all of which exhibit the signs of left hypertrophy in marked degree. Illustrations of the axis rotations are tabulated in Tables IV and V; in each case there is a conspicuous and steady anticlockwise movement.

* Lewis, 'Heart,' vol. 5, p. 367 (1914).

† Considering the conformation of the dextrogram and levogram in the heart of the dog, and the customary differences between these curves and those of the human subject and monkey, the curves of preponderance in one or other ventricle in the dog will not be expressed in quite the same fashion. I venture to predict that, in the dog, preponderance of the left ventricle will show itself, as a rule, by a deepening of *S* in all leads, but especially in Lead *II*, and to a less extent in Lead *III* (less often the changes will be the same as those found in man), and that preponderance of the right ventricle will show itself as an accentuation of *R* in all leads, but especially in Lead *II*, and to a less extent in Lead *III*.



FIGS. 4 and 5.—Two charts of the initial phases of the electrocardiograms taken from patients suffering from aortic disease, and exhibiting the signs of left ventricular preponderance. Ordinates and abscissæ as in fig. 1. The actual curves of Chart 4 are shown in Plate 21, fig. 12; these curves are somewhat exceptional, possessing features which probably indicate extreme left preponderance. The method of orientating the curves is illustrated by Plate 21, fig. 10. The standard curve is taken from a constant chest wall lead, the copper electrodes being attached by a stiff and well salted paste. The values of the charted curves are given in Tables IV and V.

TABLE IV.—Aortic Disease. (Conspicuous Left Preponderance.)

	I.	II.	III.	Angle.
sec.				
0·0050	−0·5	0	0·5	150°
0·0100	−2	0	2	150
0·0150	−4	1	5	139
0·0200	−6	2	8	136
0·0250	−6	3	9	131
0·0300	−2	5	7	106
0·0350	7	9 10	3	45
0·0400	17	17	0	30
0·0450	30 29	25	−4	22
0·0500	40 38	30	−8	19
0·0550	45	28	−17	8
0·0600	43	13	−30	−13
0·0650	38	2	−36	−27
0·0700	33 32	1	−31	−29
0·0750	29 28	3	−25	−24
0·0800	23 20	3	−17	−22
0·0850	15 13	3	−10	−17
0·0900	7·5 6·5	2	−4·5	−13
0·0950	2	0·5	−1·5	−16

TABLE V.—Aortic Disease. (Left Preponderance.)

	I.	II.	III.	Angle.
sec.				
0·0100	−2	0	2	150°
0·0150	−2	0	2	150
0·0200	−1	1	2	120
0·0250	1	3	2	71
0·0300	6	7·5	1·5	41
0·0350	14	15	1	33
0·0400	25	24	−1	28
0·0450	35 35·5	30·5	−5	23
0·0500	43 45	38	−7	22
0·0550	47 48	40	−8	21
0·0600	52 51	33	−18	9
0·0650	47 46	17	−29	−9
0·0700	29	12	−17	−6
0·0750	11 9	3	−6	−11
0·0800	3	2	−1	?11

In the case of right preponderance, the rotation of the axis should resemble the rotation of the dextrogram. It should be clockwise. I have charted the curves in the two instances of mitral stenosis, which exhibit the signs of right preponderance (fig. 6 and Plate 21, fig. 13). The axes are tabulated in Tables VI and VII; in both there is a conspicuous clockwise rotation.

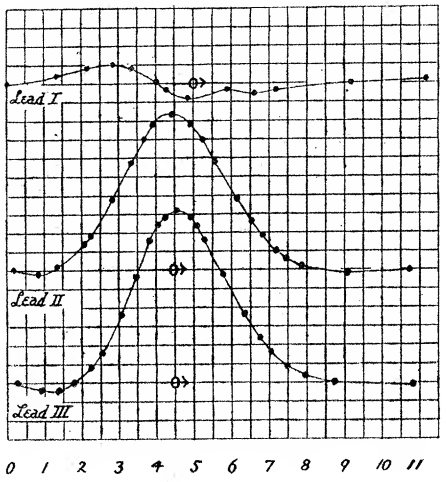


FIG. 6.—A chart of the initial phases of the electrocardiograms from a patient who suffered from mitral stenosis and exhibited the signs of right ventricular preponderance. Ordinates and abscissæ as in fig. 1. The values of these curves are given in Table VI, and the actual curves in Plate 21, fig. 13. The orientation was carried out by the method described in the explanation of figs. 4 and 5 in this and succeeding charts.

TABLE VI.—Mitral Stenosis. (Considerable Right Preponderance.)

	I.	II.	III.	Angle.
sec.				
0·0050	0·5	−0·5	−1	−60°
0·0100	1	−1	−2	−60
0·0150	2·5 3·0	1·5	−1·5	0
0·0200	4	6	2	49
0·0250	5	13	8	68
0·0300	5	23 22	17	77
0·0350	2	32 31	29	87
0·0400	0	40·5	41 40·5	90
0·0450	−3	42	46 45	93
0·0500	−4	38	43 42	95
0·0550	−3	30	33	95
0·0600	−2	22	24	94
0·0650	−3	13	16	100
0·0700	−2	7	9	102
0·0750	−1·5	2·5	4	112
0·0800	−1	1	2	120
0·0850	−0·5	0	0·5	150

TABLE VII.—Mitral Stenosis. (Right Preponderance.)

	I.	II.	III.	Angle.
sec.				
0·0050	1	0	−1	−30°
0·0100	3	0	−3	−30
0·0150	6	1	−5	−21
0·0200	7·5 7·0	5	−2	14
0·0250	8	10	2	41
0·0300	10·5	16 16·5	6	51
0·0350	11	22	11	60
0·0400	10	26·5 27	17	69
0·0450	6	26 24·5	18·5	76
0·0500	−2	12	13 14	98
0·0550	−10	−2	8	161
0·0600	−10	−7	3	?−167
0·0650	−7 8	−7	1	?−157
0·0700	−4 4·5	−4	0·5	?−156
0·0750	−2	−2	0	?−150

It is also noteworthy that while in the human levogram *Q* appears and is conspicuous in Lead *I* only, so in curves of left preponderance *Q* is maximal in the same lead and is, as a rule, confined to it; *Q* favours Leads *II* and *III* in curves of right preponderance; considering that it is absent from these leads where levograms are concerned but present in them where many bigrams are concerned, it is probable that *Q* appearing in the bigram in Leads *II* and *III* belongs to the dextrogram.*

* Having but a solitary example of dextrograms, the presence or absence of *Q* in Leads *II* and *III* of these curves cannot be decided conclusively. There appears to be a *Q* in Lead *III* of fig. 14, but the curves are tremulous and I have not charted it.

In brief, so far as the preliminary phases are concerned, there is between the levogram and curves of left preponderance on the one hand, and between the dextrogram and curves of right preponderance on the other hand, a very close resemblance. In a paper written conjointly with GILDER some three years ago* I gave the amplitude of deflections in a large series of curves taken from normal subjects. Our curves were grouped, so that those which tended to show the signs of right and left preponderance respectively, were collected together. In the first group (Cases 1–25) of our tables were those curves inclining to show right preponderance. The average values of Q in these 25 patients were as follows:—

In Lead *I*	0·2 mm.
In Lead *II*	1·0 ,,
In Lead *III*	1·2 ,,

In the third group (Cases 38–52) of our tables were those curves inclining to show left preponderance. The average values of Q in these 15 patients were:—

In Lead *I*	0·95 mm.
In Lead *II*	0·4 ,,
In Lead *III*	minute.

This analysis confirms the view that Q of the human electrocardiogram may arise in one or other ventricle. If conspicuous in Lead *I*, it is of left ventricular origin; if conspicuous in Leads *II* and *III*, it is probably of right ventricular origin. If conspicuous in all leads, it probably originates partly in each ventricle.

A Further Comparison.—In the preceding paragraphs, the curves of hypertrophy and of aberrant contractions have been compared, and close points of resemblance between them have been noted. In the fully developed types, and so far as the preliminary phases are concerned, the resemblance is almost, if not quite, complete; witness the examples placed side by side in Plate 21, figs. 11 and 12.

The origin of the two types is similar in that in each there is a lack of normal balance; in one case the result of the time factor, the precedence of one ventricle; in the other case to the factor of mass. How then may the two types be distinguished? They may be distinguished by their terminal phases. We have seen it to be the rule that in a bundle branch lesion, the curve as a whole is broadly diphasic; in Leads *I* and *III*, the terminal and prominent phase T' being opposite in sign to the chief phase, R' or S' as the case may be. In the curves of hypertrophy, T' does not show this prominence and its direction is not governed in the same manner. Its direction is the direction presented by normal curves, *i.e.*, it is usually upright, though it shows the same variations in amplitude and direction which normal electrocardiograms exhibit. That the distinguishing mark should be found in the latter phases is to be anticipated, for when the excitatory process passes by a single

* 'Phil. Trans.,' B, vol. 202, p. 351 (1912).

bundle division the ultimate spread to the opposite ventricle is along abnormal routes; the order in which the excitatory process subsides is equally abnormal. On the other hand considerable divergence from the normal order of spread in the case of hypertrophied hearts is hardly conceivable.

A hitherto unnoticed feature of the curves of hypertrophy requires brief discussion. Aberrant curves are remarkable for the duration of their initial phases. Thus in eight normal electrocardiograms the average duration of the initial phases was 0·0784 sec. (Table VIII); in seven unselected examples of right branch defects the initial phases showed an average duration of 0·1396 sec. The initial phases in curves of hypertrophy also show an increased duration, though this is only so where left preponderance is present; the average duration in six cases being 0·1005 sec. It is to be expected that in hypertrophy the spread of the excitatory process will suffer delay, for the layer of muscle to be penetrated is thicker. The reason why the increased duration is confined to hypertrophy of the left ventricle is probably two-fold: firstly, the excess of muscle is greater in left as opposed to right ventricular hypertrophy and the actual increase in muscle thickness is greater; and, secondly, the end of the initial phases of the normal curve (*S* in Leads *II* and *III*) is written by spread in the left ventricle.

TABLE VIII.—Duration of *QRS* Group (in Lead *II*).

Normal.		Left preponderance.			Right preponderance.			Right branch defects.	
Heart rate.	*QRS* group.	Lesion.	Heart rate.	*QRS* group.	Lesion.	Heart rate.	*QRS* group.	Heart rate.	Initial deflections.
	sec.			sec.			sec.		sec.
66	0·0644	Aortic	117	0·0703	Mitral stenosis	119	0·1037	85	0·1721
75	0·0736	,,	118	0·1051	,,	115	0·0738	109	0·1477
97	0·0800	,,	117	0·0848	,,	116	0·0730	92	0·1488
83	0·1038	,,	116	0·1591	,,	65	0·0639	112	0·1225
83	0·0692	,,	111	0·0709	,,	66	0·0625	85	0·1308
86	0·0956	Chronic nephritis	120	0·1129	,,	72	0·0958	90	0·1356
60	0·0782							71	0·1199
75	0·0623								
Averages.									
78	0·0784		116	0·1005		92	0·0788	92	0·1396

Normal Human Curves.

Variations occur in the forms of the electrocardiograms taken from healthy subjects; in some the curves in the three leads incline to the picture of right or left preponderance, as has been stated; other curves are transitional. I have

analysed the curves in six healthy subjects and reproduce illustrative charts in figs. 7, 8, and 9 and the values in Tables IX, X, and XI. In instances (fig. 7 and Table IX) where *R* is prominent in all leads, but chiefly so in Lead *II*, and where *S* fails to appear in Leads *II* and *III*, a simple and uninterrupted clockwise movement of the axis is demonstrated. The rotation of the axis is controlled by the right ventricle throughout. Nevertheless these curves may fail to show the predominance of *R* in Lead *III*, as compared to *R* in Lead *I*. On the other hand, they do show *Q* to be confined to Leads *II* and *III*, and *S* to be confined to Lead *I*. They bear several traces of the right preponderance in their outlines and amplitudes but do not exhibit the complete series of signs.

A curious and interesting combination of dextrogram and levogram is illustrated by fig. 8 and Table X. The first phase *Q* is present in Lead *I* only, and the early rotation is anticlockwise and controlled by the left ventricle. During the inscription of *R* in its greater part, the rotation alters its direction and moves definitely but very slightly in a clockwise fashion; over this period the left and right rotations are almost balanced, the right prevailing; finally, during the inscription of *S* the original anticlockwise movement is resumed, the left rotation prevailing. These curves are from an instance of very slight left preponderance within the bounds of normality.

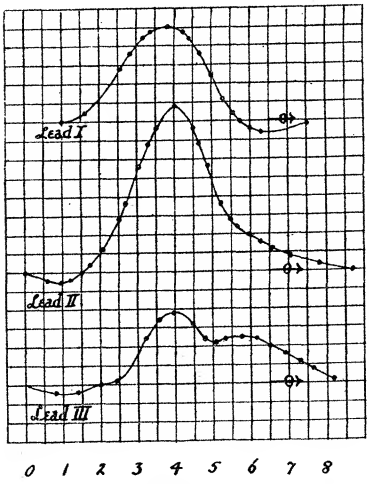


FIG. 7.

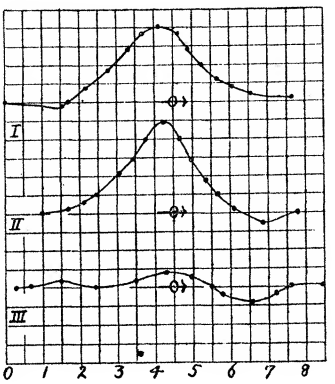


FIG. 8.

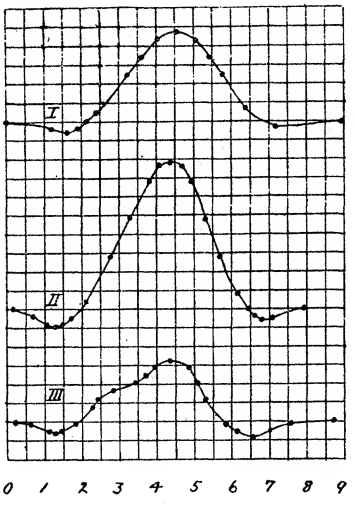


FIG. 9.

FIG. 7.—Chart of the initial phases of a normal human electrocardiogram exhibiting a tendency towards right ventricular preponderance. Ordinates and abscissæ as in fig. 1. The values are given in Table IX.

FIG. 8.—Similar chart from a normal subject showing a very slight left preponderance. At the beginning and end the levogram prevails. In the central part of the chart the dextrogram is predominant.

FIG. 9.—Similar chart from a normal subject showing some preponderance of the left ventricle. At the beginning of the chart the dextrogram prevails, but the remaining rotation is governed by the levogram.

In normal hearts, and while *R* is written and this deflection is prominent in Lead *III*, a clockwise rotation always prevails, indicating predominance of the dextrogram. The regularity of this movement may be disturbed for short periods either at the beginning or at the end of the chart, where the left or anticlockwise rotation asserts itself. Lastly, some normal human curves, which incline towards left preponderance, show an almost uninterrupted anticlockwise rotation of the axis. An example is shown in fig. 9 and Table XI.

TABLE IX.—Normal.

	I.	II.	III.	Angle.
sec.				
0·0050	0	−1·5	−1·5	−90°
0·0100	0	−2·5	−2·5	−90
0·0150	1·5	0	−1·5	−30
0·0200	6 5·5	5·5	0	30
0·0250	13	14 14·5	1·5	35
0·0300	20	27 28	8	46
0·0350	24	38 40	16	53
0·0400	25	44	19	55
0·0450	21	37·5 37	16	55
0·0500	12	23	11	59
0·0550	2·5	14 14·5	12	81
0·0600	−2	10	12	99
0·0650	−3	6·5	10 9·5	108
0·0700	−2·5	4·5	7·5 7·0	111

TABLE X.—Left Preponderance within the bounds of normality.

	I.	II.	III.	Angle.
sec.				
0·0100	−1	0	1	?150
0·0150	−1	1	2	120
0·0200	2·5	2·5 3	0·5	39
0·0250	6·5 6	6	0	30
0·0300	11 10	10·5	0·5	32
0·0350	16	16 18	2	36
0·0400	20	23 23·5	3·5	38
0·0450	19	23	4	39
0·0500	12·5	14 15	2·5	39
0·0550	7	7	0	30
0·0600	5	2	−3	−70
0·0650	2·5	−1·5	−4	−52

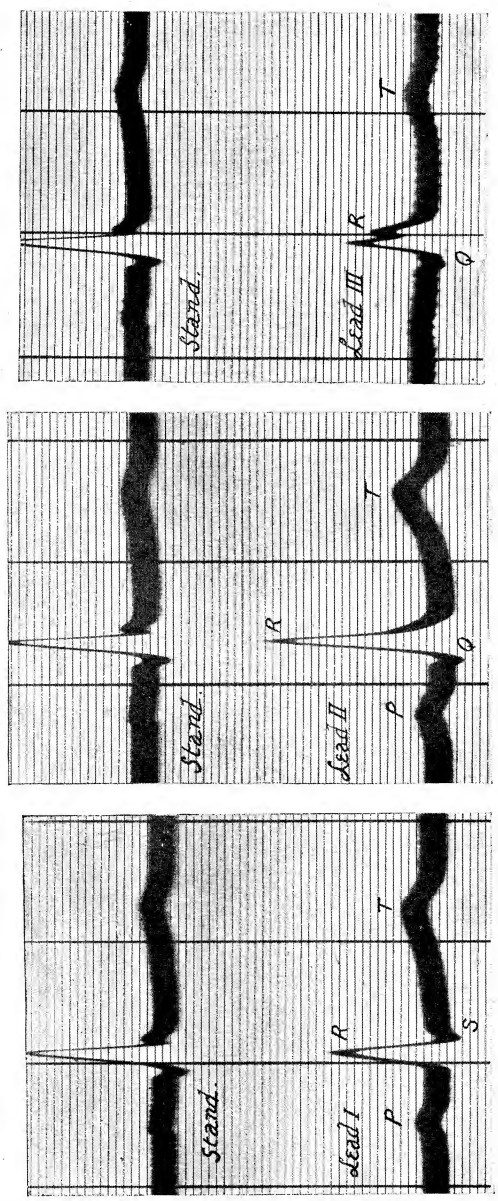


FIG. 10.

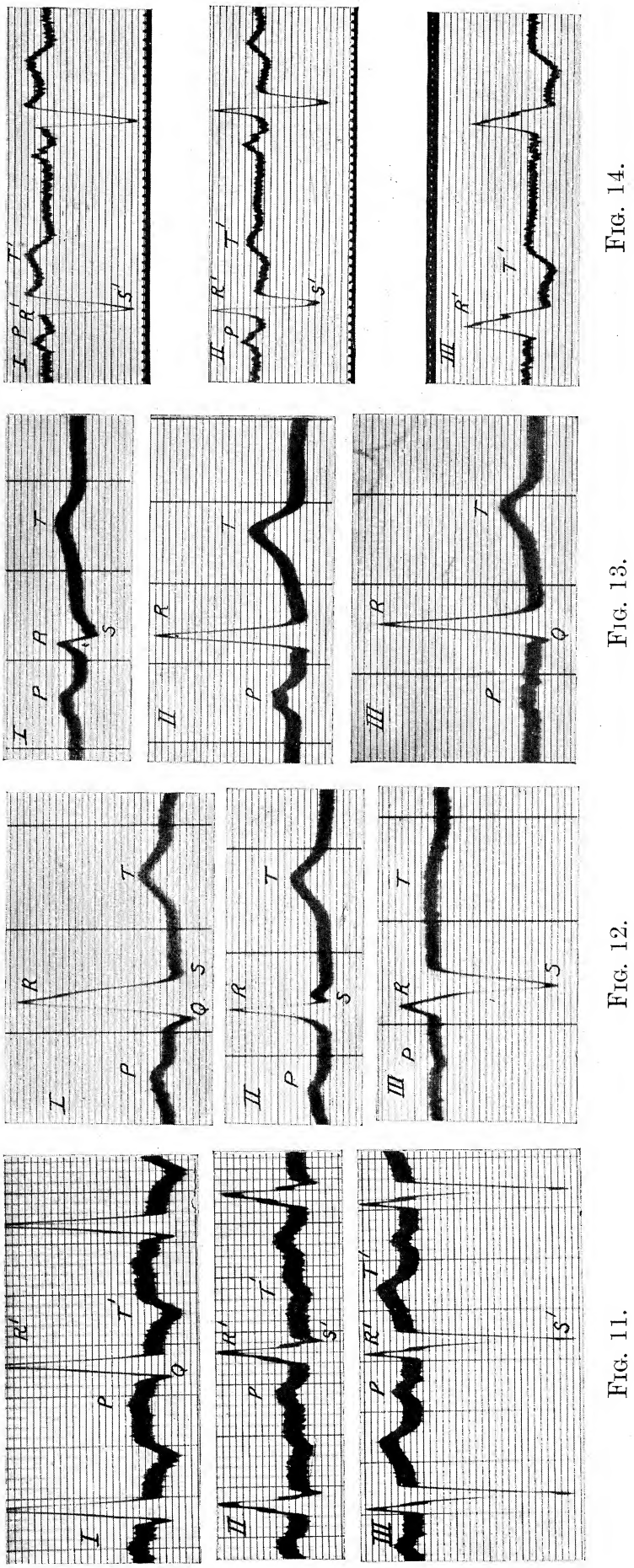


FIG. 14.

FIG. 13.

FIG. 12.

FIG. 11.

TABLE XI.—Left Preponderance just within the bounds of normality.

	I.	II.	III.	Angle.
sec.				
0·0050	−0·5	−1	−0·5	−120°
0·0100	−1	−3·5 3	−2	−109
0·0150	−2·5	−4 4·5	−2	−124
0·0200	−1	0	1	150
0·0250	3	9 9·5	6·5	72
0·0300	9	18·5 18	9	60
0·0350	16	28 27	11	54
0·0400	21·5	37 36·5	15	55
0·0450	23·5	39 39·5	16	53
0·0500	21·5	33	11·5	50
0·0550	16	19	3	38
0·0600	9	7	−2	18
0·0650	2	−1·5 2	−4	−60
0·0700	−0·5	−3	−2·5	−101

EXPLANATION OF PLATE 21.

Fig. 10.—Three photographs from a normal human subject. The upper curve in each is the standard lead, a fixed lead from the chest wall. The lower curves are from Leads *I*, *II*, and *III*. Ordinates, 1 cm. = 1 millivolt; abscissæ, 0·2 sec. These curves were used in constructing the chart of fig. 7.

Fig. 11.—Electrocardiograms from the three leads, indicating a right bundle branch lesion. Ordinates, 1 cm. = 1 millivolt; abscissæ, 0·2 and 0·4 sec. These curves were used in constructing the chart of fig. 1.

Fig. 12.—Three electrocardiograms from a patient suffering from aortic disease, and published for comparison with those of fig. 11. The resemblance is close, so far as the initial phases of the two sets of curves are concerned. Ordinates, 1 cm. = 1 millivolt; abscissæ, 0·2 sec. Used in constructing the chart of fig. 4.

Fig. 13.—Three electrocardiograms from a case of mitral stenosis. Ordinates and abscissæ as in fig. 12. Used in constructing the chart of fig. 6.

Fig. 14.—Three electrocardiograms from a patient in whom a defect in the left bundle branch is supposed to have existed. Ordinates, 1 cm. = 1 millivolt. Time marker in 0·03 sec. Used in constructing the chart of fig. 2.

Part V.—*The Bird's Heart.*

[PLATE 22.]

The spread of activity in the bird's ventricle has received no attention hitherto. We know, from the sequence of chamber contraction, that the ventricle receives its impulse from the auricle; the path of the spread is still imperfectly understood. The minute anatomy of the auriculo-ventricular junction was examined by TAWARA* who expressed his belief in the existence of a muscular connection between the two chambers. MCKENZIE and ROBERTSON† stated in 1910 that they could find no specialised musculature at the $A-V$ junction of birds; but described a thin leash of cardiac muscle connecting the right auricle with the right ventricle at the right and posterior part of the groove, in the region where the left superior cava has its outlet. KULB's description‡ differs somewhat from that of these writers; he finds a tract of tissue, in its appearance resembling the bundle of His in mammals, running across the junction at the posterior aspect of the heart, from a point where the right and left auricular walls fuse, and being prolonged forward and downwards to join the musculature of the ventricular septum.

That a muscle tract exists in this neighbourhood seems clear, but its destination is obscure. Its termination in a ventricular arborisation comparable to that of the mammalian heart has not been described. Nevertheless, fibres of very similar constitution to those known as the fibres of Purkinje in the mammalian ventricle have long been known to exist in the bird's heart (OBERMEIER, HOFMANN§). The general distribution of these fibres is also known: they are found subendocardially in the ventricle; but there is no subendocardial concentration, such as is found in the mammalian heart; large collections of the same cells occupy deep positions in the heart wall, especially in the neighbourhood of arterial branches (HOFMANN, TAWARA, MCKENZIE and ROBERTSON); these cell collections are indeed conspicuous in sections of the bird's ventricle; the detailed architecture of the columns of cells, if a connected architecture is present, remains untraced.

FLACK's experimental work‖ tends to confirm the existence of a functional union between auricle and ventricle on the posterior aspect of the groove, for he states that

* 'Das Reizleitungssystem,' etc., Jena, 1906.

† 'Brit. Med. Journ.,' vol. 2, p. 1161 (1910).

‡ 'Proc. Internat. Congress,' London, Section III, p. 92 (1913).

§ OBERMEIER, 'Archiv f. Anat., Physiol. u. wissens. Med.,' pp. 245 and 358 (1867); HOFMANN, 'Zeitschr. f. wissens. Zool.,' vol. 71, p. 486 (1902).

‖ 'Archiv. Internat. de Physiol.,' vol. 11, p. 120 (1911).

ligatures transfixing the groove in this region produce disturbances of sequence,* and that ligation of other parts of the junction is without effect. These experimental observations still need to be confirmed and extended; especially is it desirable that the experimental lesions should subsequently be explored microscopically.

Method.

The method adopted in the present researches has been a continuation of that employed upon other vertebrata. Fowls and pigeons and also a swan have been anæsthetised with ether; in some instances the animals have been subsequently killed by pithing, respiration being in all instances maintained artificially. The heart has been exposed by a ventral route, the pectoral muscles being disconnected from the sternum, and the latter split and removed. In exploring the ventral surface of the ventricles, the standard curve has been a lead from the right wing to the left thigh. In exploring the lateral and dorsal surfaces, it has been necessary to displace the heart; in these circumstances care has been taken to maintain good tissue contact between the heart apex and the liver or lung, and the electrodes of the standard lead have been adjusted to parts of the body wall in line with the new heart axis. In this manner, and only in this manner, can the usual type of standard curve be maintained.

Direct leads have been taken, one contact lying on the surface of the heart, the other being attached to the left chest wall. Accurate projected or measured drawings have been made of the heart *in situ* in all cases. A single surface only of the heart has been investigated in a given experiment, the standard curve thus remaining quite constant.

The bird's axial electrocardiogram in extracardial leads differs materially in outline from an axial mammalian electrocardiogram; it consists in the main of a prominent downstroke S (Plate 22, fig. 13, etc.), followed by a prominent upright and slow deflection T. S is almost always preceded by a diminutive upward spike R. In using the axial curve as a standard, I adopt the summit of R (beginning of the downstroke S) as the safest point for measurement. The time distance between this point and the onset of the ventricular deflections in the same lead is also measured.

Surface Distribution.

The *ventral surface* is exemplified in the natural size outlines of a swan's heart (fig. 1), a fowl's heart (fig. 2), and a pigeon's heart (fig. 3). The excitation wave first appears in the region of the ventral attachment of the septum (the broken line in these and subsequent figures indicates the boundary of the right ventricular cavity, or the limit of the septal attachment on the right side) in its lowest

* Conclusions in respect of A–V conduction in the bird must be drawn circumspectly, considering how easily heart-block follows manipulation of the bird's heart and its peculiar susceptibility to adverse experimental conditions.

or apical two-thirds. The readings of the excitation wave traced from this septal line towards the base of the right ventricle increase in quantity; there is with considerable, though not absolute, uniformity a delay in the appearance of surface activity as the base is approached. At the same time, the quantitative differences are small, the whole ventral surface of the right ventricle becoming active

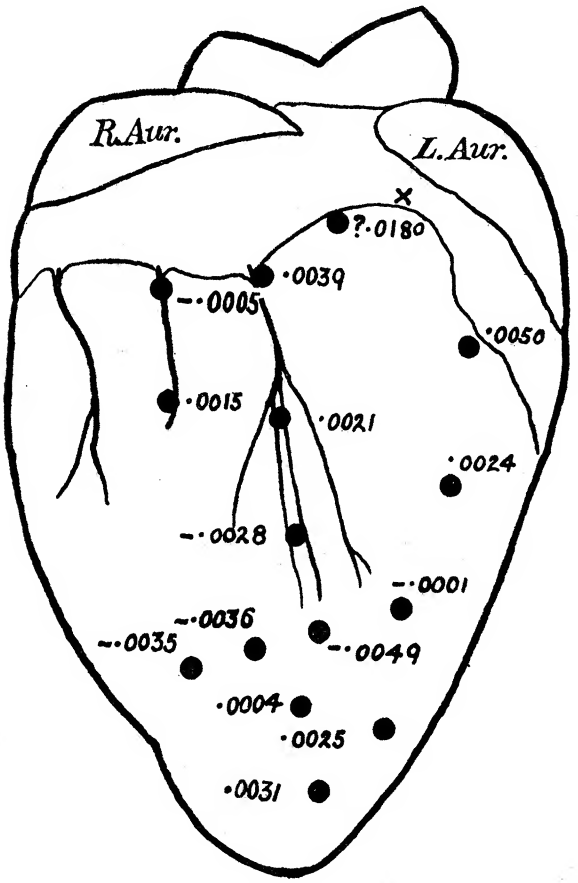


Fig. 1.

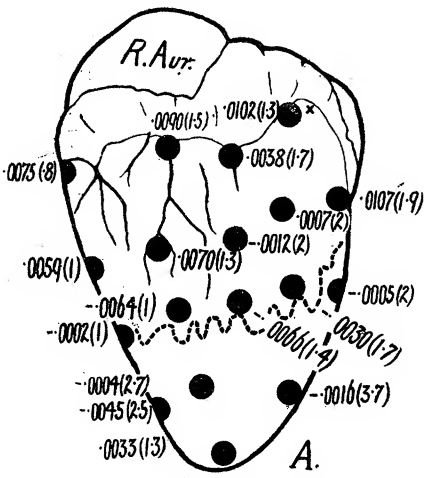


Fig. 2.

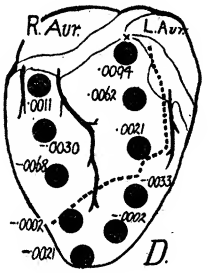


Fig. 3.

Fig. 1.—A natural-size outline of the ventral surface of a swan's heart, showing a number of superficial readings. In this and succeeding figures the size of the contacts is represented as accurately as possible. The conus is marked × in this and subsequent figures.

Fig. 2.—A natural-size projected outline of the ventral surface of a fowl's heart (Fowl *A*), showing a number of surface readings. The broken line in this and succeeding figures represents the limits of the right ventricular cavity.

Fig. 3.—Natural-size projection of the ventral surface of the heart of Pigeon *D*, showing surface readings.

within $1\frac{1}{2}$–2 hundredths of a second. In brief, the distribution, so far as it has been described, is generally very similar to that presented by the heart of a dog.

If the readings are traced from the attachment of the septum towards the apex, a similar arrangement is discovered; again there is delay. Thus the septal region stands as the earliest to be activated, the right and left walls on either side of it are

activated later. The latest point to become excited has been, with a solitary and unimportant exception, the conus.

The distribution over the *dorsal and right surfaces* of the ventricle is illustrated by the diagrams of two fowls' hearts (figs. 4 and 5) and a pigeon's heart (fig. 6). Though the distribution in the several diagrams lacks uniformity it may be stated, as a general rule, that as we move along the posterior attachment of the septum from $A-V$ groove towards the apex of the right ventricle, the readings become earlier; in isolated instances, it is true, they appear to remain almost unaltered. Of this, as of the ventral surface, the broad statement is also true that the values rise as they are traced from septum to left apex. Speaking with all my material in mind, it is the general rule that, as readings are traced along the $A-V$ groove from dorsal septal attachment to conus region, the values rise. It is especially to be remarked that the point which overlies the junction of posterior septal attachment and the fat of the $A-V$ groove is often relatively late, and is never the earliest point of surface distribution.

The left and left dorsal surfaces are illustrated in the diagrams of two fowls' hearts (figs. 7 and 8) and of a pigeon's heart (fig. 9).

They again show the relative delay at the $A-V$ groove, a lesser delay at the heart's apex, while activity first appears in the region of the septal attachments, and especially in the mid-regions of the heart. This aspect of the heart illustrates especially the left ventricle, and the approximation of all its ventricular readings must be emphasised; the whole of its surface is generally covered in about a hundredth of a second.

The Ventricular Surface as a Whole.—In considering the distribution of the surface as a whole, we are at first struck by its general resemblance to that observed in the dog. It resembles the surface distribution in the last-named animal in a particular respect. Many points are supplied simultaneously, and this is especially so over the left ventricle; as is the case over the surface of the dog's heart, the relative values of the readings exclude conduction along muscle bands; where the readings appear in succession the spread has a rapidity which is too great, and the other regions lack the element of uniform succession. It is impossible to review the surface distribution here and in the dog without concluding that the spread is from within* and that the excitatory process is conveyed to the surface by a large number of distinct channels. The chief channel is evidently the septum, for much more often than not the value at a given surface point is great according as it is removed from a septal attachment.

* A conclusion which is supported by comparing the intrinsic readings with those of the commencing axial curve and the extrinsic deflections. The axial curve begins in a short upstroke, R, which precedes S by approximately 0·0100 sec. The earliest extrinsic deflections precede S by values varying from 0·0100 sec. to 0·0150 sec. These early deflections are to be explained as resulting from the excitation of the deeper musculature.

I conclude from surface distribution alone that the first spread is to the septum, and that the general spread is fanwise, along radiating paths in the septum, and from its attachment over the free walls of the ventricles. But that the spread is

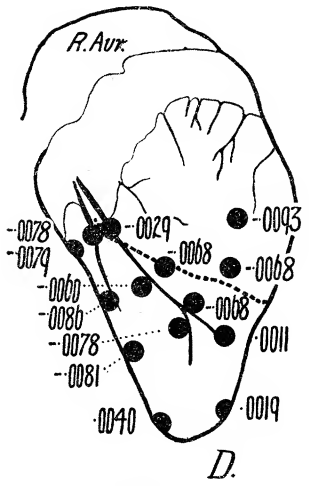


FIG. 4.

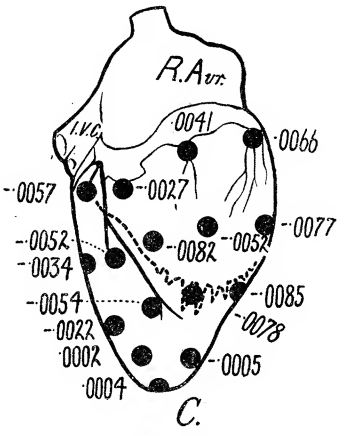


FIG. 5.

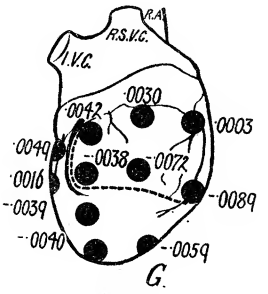


FIG. 6.

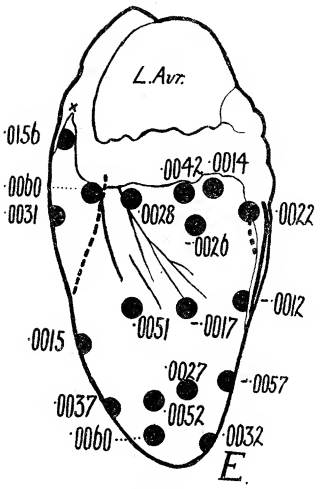


FIG. 7.

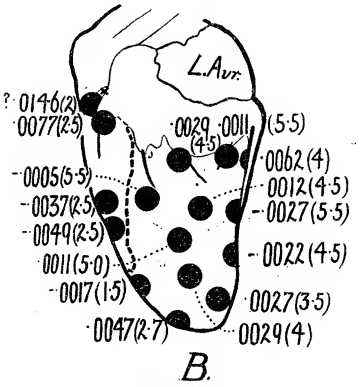


FIG. 8.

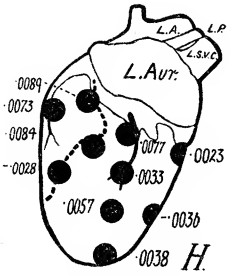


FIG. 9.

FIGS. 4 and 5.—Two natural-size projected outlines of the hearts of Fowls *D* and *C*. Seen from the right side and showing surface readings.

FIG. 6.—Natural-size projected outline of the heart of Pigeon *G*. Seen from the right side and showing readings. *I.V.C.* = inferior vena cava; *R.A.* = right aorta.

FIGS. 7 and 8.—Two natural-size projected drawings of the hearts of Fowls *E* and *B*. Seen from the left side and showing surface readings.

FIG. 9.—Natural-size projected drawings of the heart of Pigeon *H*. Seen from the left side and showing surface readings.

through muscle elements cannot be entertained, so it seems to me, on account of its rapidity; it is through a special distributing system of fibres, as in the dog.

The surface distribution in the bird differs, however, in a noteworthy respect from

that discovered in the dog. It differs in that the values over the left ventricle are extraordinarily low. In the dog, as the septum is passed in travelling from right ventricle to left over the ventral surface, the values rise abruptly by some two hundredths of a second or more, and are maintained over the whole left ventricular surface except at the extreme apex. The high values over the left ventricle, as ROTHSCHILD and I were able to show, are the result of muscle thickness and of slow conduction in this muscle, as compared to the conduction rate in the distributing fibres. But in the bird's heart the disproportion in wall thickness between right and left ventricles is much greater than in mammals. How, then, are the low readings to be explained? They are to be explained by an observed peculiarity of the bird's ventricle which accords remarkably with my general hypothesis of the distribution; fibres of Purkinje type penetrate the walls of the ventricle and lie deep in its substance. It is true that occasional strands of the same kind are to be seen in the dog's heart, but the penetration is neither so deep nor so widespread. If, as it is reasonable to conclude, the peculiar fibres of the bird's ventricle, which so closely resemble the Purkinje fibres of the mammalian heart, subserve the same function, then the surface distribution in the bird's heart is not difficult to explain. The reading at a given point on the surface is controlled in the dog by two factors, the length of the Purkinje tract concerned, and the thickness of the underlying muscle. In the bird, I believe that the first factor alone appreciably controls the reading; and, speaking generally, while the value of the reading varies, whether over left or right ventricle, according as the corresponding point lies near or at a distance from the septal attachment, so we may conjecture, if we may not conclude with a reasonable degree of certainty, that the excitation wave spreads by special tracts from above downwards in the septum and through similar special tracts over the free ventricular walls.

Rates of Conduction.

The presence of a special system of distributing fibres is confirmed by the experiences of stimulation and the rate at which an artifically propagated wave of excitation is conducted over various regions of the heart.

Working in the same manner as upon the mammalian heart, I have tested the rate of conduction over right ventricle, left ventricle and septum in a number of birds. In the dog, it will be remembered, the rate of conduction varies, according to the thickness of the muscle layer which overlies the Purkinje network. On the right ventricle rapid, on the left it is slow; for in the former case conduction is chiefly by Purkinje substance and in the latter case by the more slowly conducting muscle. In the bird's heart, where a subendocardial system of conducting fibres is not conspicuous and penetration of the musculature is almost universal, a difference in the conduction rate over the two ventricles is not anticipated. Moreover, it is not found. (See Table I.) The rates of conduction vary considerably, but are high, ranging from 800 to 6000 mm. and more per second. They are no greater over right than over left

TABLE I.

	Right ventricle.			Left ventricle.			Across septum.		
	Interval.	Distance.	Rate of transmission.	Interval.	Distance.	Rate of transmission.	Interval.	Distance.	Rate of transmission.
	sec.	mm.	mm./sec.	secs.	mm.	mm./sec.	secs.	mm.	mm./sec.
Swan A .	0·0063	17·0	2540	0·0020	13·0	6500	0·0232	25·5	1099
Fowl A .	0·0034	13·0	3823	−0·0014	12	∞	0·0186	13·5	726
Fowl D .	0·0104	11·5	1106	0·0089	11	1236			
				0·0187	12	641			
Fowl E .	0·0089	10·0	1123	0·0125	10	800	0·0159	11·0	670
Fowl F .	0·0100	12·75	1275	0·0092	10·0	1087	0·0228	15·0	658
	0·0083	10·00	1205	0·0143	16·0	1119	0·0154	14·0	909
Fowl G .	0·0135	12·00	889	0·0117	13·0	1111			
	0·0107	12·00	1121	0·0111	13·0	1170			

ventricle, and present similar variations over the two chambers. It occasionally happens that with two contacts in line with the point of stimulation, the distal contact first receives the excitation wave, an observation which was made upon the left ventricle of a fowl. This phenomenon has never been encountered in the dog except over the right ventricle; it is attributed in both instances to the presence of a favourable Purkinje channel of conduction to the distal contact. The rates across the septum in the bird are uniformly low, as in the dog's heart, suggesting that, to some extent at least, the supply of right and left ventricle is separate, as it unquestionably is in the mammal.

THE ELECTRICAL AXIS AND THE MEANING OF THE AXIAL ELECTROCARDIOGRAM IN EXTRACARDIAL LEADS.

Records have been taken from two fowls and two pigeons, using pairs of contacts arranged as a triangle. Non-polarisable contacts were placed beneath the skin at the base of each wing; a third contact was placed immediately below and deep to the free end of the breast-bone in the middle line. The curve yielded by each lead (*I*, *II*, *III*) from a pair of contacts was taken simultaneously with a standard curve from a contact at the base of the neck and right chest wall. Subsequently, and after washing out the blood, formalin was injected into the vessels and the heart hardened *in situ* without breaking the chest wall, the bird being then cut in the plane of the electrodes. The diagrams which accompany the charted curves accurately represent the relations of the hearts to the contacts; the arrows and numerals indicate

the direction and numbers of the leads. The angles expressing the electrical axis are related to the base line of Lead *I*.

The electrical axis of the bird's heart generally moves, during the initial phases of the activity, through approximately 180°. For a first and short phase of about 1/100 second the axis is in the line of the body and is directed downwards, indicating a general movement of the excitation wave in this direction. These events precede the chief downward movement (*S*) in these leads and correspond to diminutive upward phases (*R*) which are the rule in axial curves from the bird's heart; they occur therefore immediately prior to the appearance of the excitation wave on the surface of the heart; or correspond, in some instances, to its earliest appearance on the surface in the region of the septal attachments.

In one of the fowls these initial phases were inconspicuous and do not figure in the corresponding Table (Table II, Fowl I). There may be, as in the other fowl (fig. 10 and Table II, Fowl H), a gradual rotation of the angle, but usually (fig. 11 and Table III) there is a more abrupt displacement, and the electrical axis turns almost at once through 180° from 90° to −90°, where it remains. In Leads *II* and *III* deep deflections (S) are the resultants, while the curve in Lead *I* remains almost

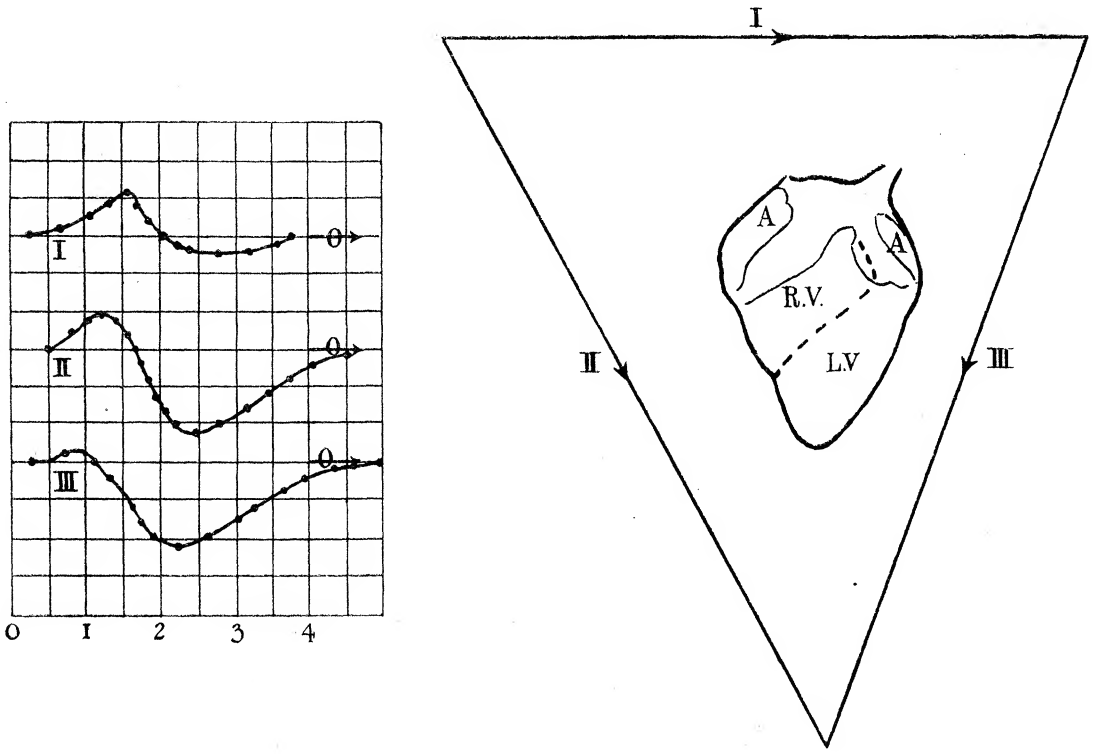


FIG. 10.—(Fowl H.) Chart of the initial phases of the electrocardiogram in Leads *I*, *II*, and *III*. Ordinates, 6 cm. = 1 millivolt; abscissæ, 1 cm. = 0·01 sec. The corresponding electrical axes are given in Table II.

And a diagram of the heart (natural size), showing its exact relation to the leads. The ventral surface of the heart was just within the plane of the contacts.

TABLE II.

	Fowl H.				Fowl I.			
	I.	II.	III.	Angle.	I.	II.	III.	Angle.
sec.								
0·0050	0·5	0	0	—	2·5	−1	−3·5	−46°
0·0080	1	2	1	60°	4	−8	−11 12	−71
0·0100	2	3	1	49	1·5	−14	−16 15·5	−85
0·0130	4·0	4·5 3·0	−1	16	−1	−17	−17 16	−93
0·0150	5·5 6·0	3	−3	0	−1	−16	−15	−93
0·0180	3·5	−3	−8 6·5	−57	−1	−13·5	−12·5	−94
0·0200	1	−7	−10 8	−83	−1	−11	−10	−95
0·0230	−1	−11	−11 10	−95	−1	−9	−8	−96
0·0250	−2	−11 12	−10	−99	0	−7 6	−6	−90

NOTE.—In this Table the potential values under columns I, II, and III are expressed in sixtieths of a millivolt. This Table and the succeeding Table have been constructed from the original charts; the corresponding published figures have been redrawn and are not precisely accurate.

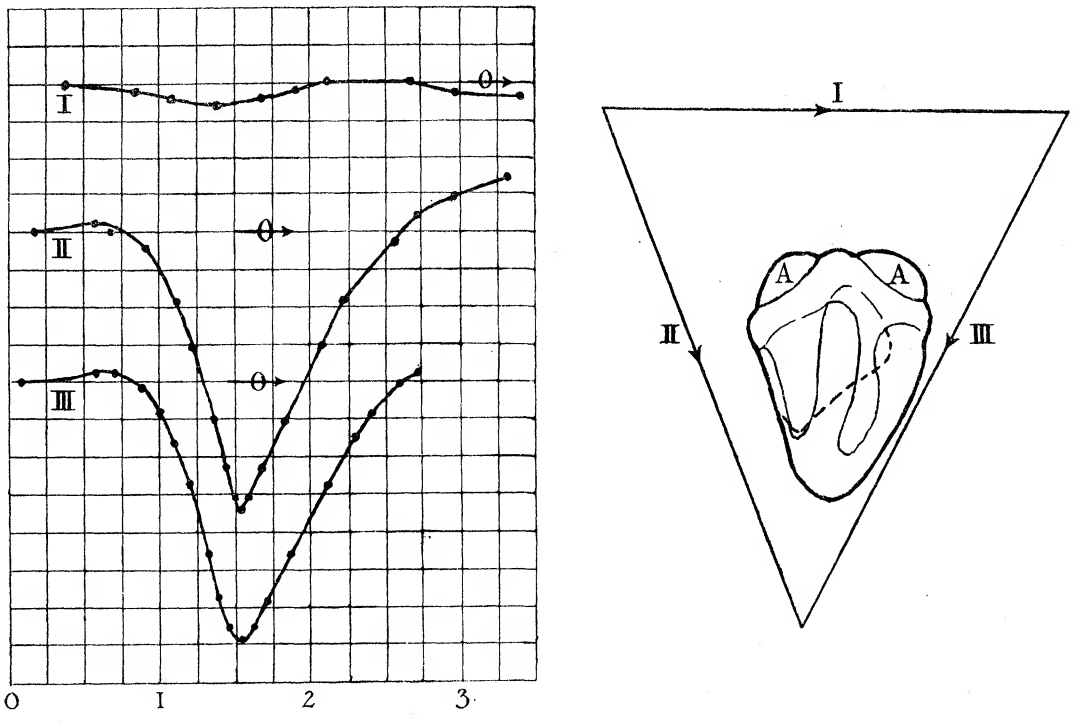


FIG. 11.—(Pigeon J.) A chart showing the initial phases of the electrocardiogram in Leads *I*, *II*, and *III*. Heart rate, 180 per minute. Ordinates, 2 cm. = 1 millivolt; abscissæ, 2 cm. = 0·01 sec. The corresponding electrical axes are given in Table III.

And a diagram (natural size) showing the heart and its exact relation to the leads. A section in the plane of the contacts opened the cavities of both ventricles, the outlines of which are shown. The broken line indicates the extreme limits of the right ventricular cavity.

TABLE III.

	Pigeon J.				Pigeon K.			
	I.	II.	III.	Angle.	I.	II.	III.	Angle.
sec.								
0·0050	0	1	1	90°	0	1·5	1·5	90°
0·0075	−1	0	1	?150	0	1	1	?90
0·0100	−1·5	−5·5 6·0	−4·5	−104	0	−16	−15 16	−90
0·0125	−2·5	−19 19·5	−17	−97	−2	−39	−37	−93
0·0150	−2·5	−36 36·5	−34	−94	−6	−63	−57	−95
0·0175	−1·5	−29	−27·5	−93	−5	−43 42	−37	−96
0·0200	−0·5	−19 18·5	−18	−91	−2·5	−19 19·5	−17	−97

NOTE.—In this Table the potential values under columns I, II, and III are expressed in 0·05 millivolt (Pigeon J) and 0·075 millivolt (Pigeon K).

isoelectric. The deflections (S in Lead *II* and *III*) speak for a general movement of the excitation wave in the upward direction in the axis of the body.

Where the movement is in short steps, the succession is regular and anti-clockwise, and the curves in Leads *I*, *II*, and *III* present a considerable resemblance to those seen in the dog, where the right bundle division is not conducting. I read the rotation of the electrical axis as expressing an alteration in the average direction in which the fibres are activated, and find that it confirms my view that the excitatory process first spreads downwards through the septum, and later upwards through the free walls. The abrupt passage from an angle of 90° to an angle of −90°, as opposed to a more gradual passage through a wider angle in the mammal, is to be explained by the arrangement of the special distributing tracts. In the mammal these are sub-endocardial, and the spread in the septal and free wall of the ventricle is away from the endocardial surface; in the bird, where the sub-endocardial concentration is not conspicuous, and where the special cells are found abundantly in the substances of the musculature, the path of the spread may be expected to be less at right angles to the wall, and more in line with it.

Thus the rotation of the electrical axis as calculated from extracardial leads from the bird's heart is very similar to that calculated from the levogram of the dog's heart. The bird's axial curve is in the main an expression of left ventricular activity. In the dog (Table IV) the average ratio of the weights of the separated right and left ventricles is as 1 is to 1·8; in the bird's heart the same ratio is as 1 is to 3·3 (Table V). The bird's axial curve differs from the levogram of the dog in several important respects however.

1. There is no preliminary downward deflection *Q*; the spread in the upper part of the septum is not chiefly from left to right or right to left as in the dog and man; it is chiefly from above downward.

2. The initial phases, *R* and *S*, have relatively a short duration (Table VI), amounting usually to 0·0300 or 0·0400 second, and constituting as a rule no more than one-fourth or one-eighth of the whole ventricular complex (in the dog they constitute approximately one-third of the full complex). The rapidity of spread through special intramural tracts is partly responsible for this shortening of the initial phases, which is also conditioned by simultaneous rather than subsequent spread through the right ventricle.

3. The axis tends to depart but little from the vertical; and this almost vertical set of the axis, as has been stated, is attributed to spread in the line of the septum or ventricular wall.

TABLE IV.—Dog's Ventricular Weights.

No.	Right.	Left.	Septum.	L/R.
	grm.	grm.	grm.	
1	22·0	46·5	11·7	2·11
2	30·5	50·0	13·0	1·64
3	25·7	43·5	10·5	1·69
4	25·0	45·5	15·5	1·82
5	12·0	25·5	7·5	2·12
6	47·5	75·0	22·5	1·58
7	22·5	37·5	14·0	1·67
8	22·5	40·5	19·0	1·80
9	53·5	92·0	28·5	1·74
			Average . . .	1·80

TABLE V.—Turkey's Ventricular Weights.

No.	Right.	Left.	Septum.	L/R.
	grm.	grm.	grm.	
1	5·2	16·5	5·8	3·17
2	4·1	12·5	4·5	3·05
3	5·1	15·6	4·6	3·06
4	4·6	12·6	4·2	2·74
5	4·6	17·5	5·4	3·80
6	2·5	9·4	4·0	3·76
			Average . . .	3·26

It is improbable that the right ventricle materially contributes to the curves; where it does contribute it contributes in the same direction as does the left ventricle. While the excitation wave is rising through the free wall of the left ventricle it is also rising through the free wall of the right ventricle. The absence of a conspicuous

R deflection in the bird is to be explained by the fact that the whole free wall of the right ventricle is activated in an upward direction.

TABLE VI.

	Average heart rate.	R.	Summit of R.	Bottom of S.	Return of S.	Latest intrinsic reading.	End of T (rate varying).	Length of intrinsic.
Pigeon C. .	221	−0·0069	0·0000	0·0094	0·0218	0·0101	0·1618	
„ D. .	280	−0·0095	0·0000	0·0121	0·0243	0·0094	0·1213	
„ E. .	233	—	—	—	—	0·0042	—	
„ F. .	179	−0·0079	0·0000	0·0123	0·0270	0·0058	0·1670	
„ G. .	213	−0·0076	0·0000	0·0130	0·0304	0·0049	0·1510	
„ H. .	167	−0·0078	0·0000	0·0122	0·0310	0·0089	0·1681	
„ I. .	105	?	0·0000	0·0160	0·0382	0·0067	—	
Swan . . .	257	−0·0060	0·0000	0·0117	0·0310	?0·0189 ?0·0050	0·1509	
Fowl A . .	122	−0·0126	0·0000	0·0132	0·0357	0·0107	0·2572	
„ B . .	132	−0·0109	0·0000	0·0133	0·0378	?0·0146 ?0·0077	0·3048	
„ C . .	161	−0·0131	0·0000	0·0105	0·0310	0·0066	0·2493	
„ D . .	210	−0·0108	0·0000	0·0131	0·0279	0·0040	0·1921	
„ E . .	155	−0·0055	0·0000	0·0118	0·0286	0·0156	0·1615	

The general hypothesis of spread in the bird's heart may be summed up diagrammatically (fig. 12). In constructing this figure I have chosen readings which

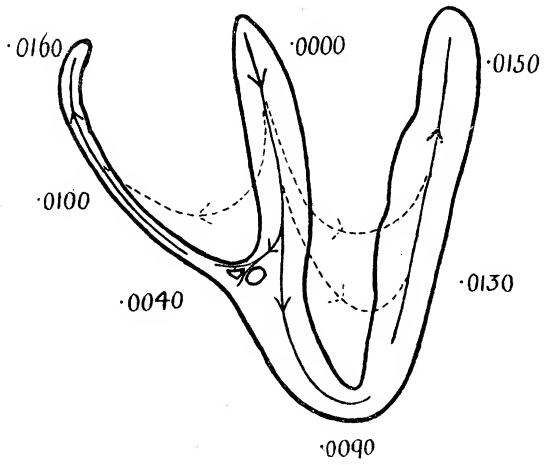


FIG. 12.

may be taken as fairly representing the regions of the heart seen in this coronal section, and have added to each 0·0100 second* to render them compatible with the plotted curves of the charts (figs. 10 and 11).

Now, the initial phases of the bird's axial curve have a duration which may be taken approximately at 0·0350 second (see Table VI). Of this time interval the first

* The distance R—S.

0·0200 second is covered by the spread of the excitation process in the ventricle. Add to this 0·0100 or 0·0150 second as the time taken for the full development of surface potential, and the sum total, 0·0300 or 0·0350 second, is in sufficiently close agreement with the length of the initial phases. The general rule, that the time interval covered by direct readings from the heart is the same as the length of the initial phases, applies to the bird's heart, as to the heart of other vertebrata.

EXPLANATION OF PLATE 22.

Fig. 13.—(Fowl C.) Simultaneous electrocardiogram from Lead *II* and curve from direct ventricular lead. Illustrating the method of obtaining direct readings from the heart. The curve from the direct lead shows a minute extrinsic and a sharp and tall intrinsic deflection. Ordinates, for Lead *II*, approximately 1 cm. = 1 millivolt ; for the direct lead 1 mm. = 1 millivolt ; abscissæ = 0·2 sec.

Fig. 14.—Similar curves from a swan. In the direct lead, the extrinsic deflection is a distinct and sharp downward deflection and this is followed by the usual and prominent intrinsic spike. Ordinates and abscissæ as in fig. 13.

Fig. 15.—(Pigeon J.) Leads *I*, *II* and *III*, each taken simultaneously with a standard curve. The latter was from the top of the sternum to the lower part of the right chest wall. Ordinates of the three leads, approximately, 1 cm. = 1 millivolt ; abscissæ = 0·2 sec.

Fig. 16.—(Fowl I.) Similar photographs from Fowl I. Ordinates of the three leads, approximately, 3 cm. = 1 millivolt ; abscissæ = 0·2 sec.

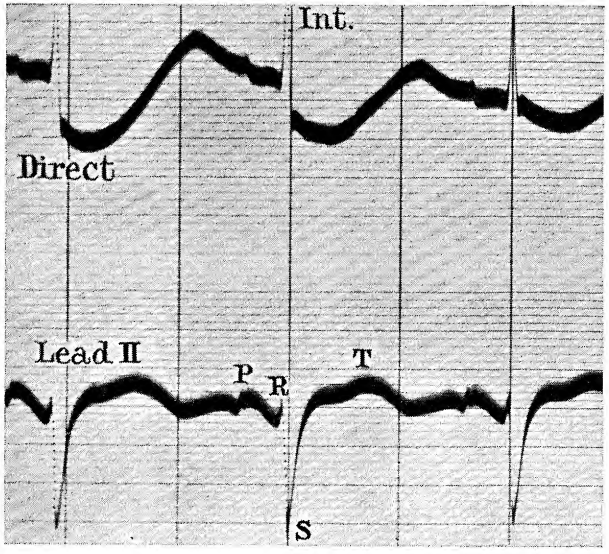


FIG. 13.

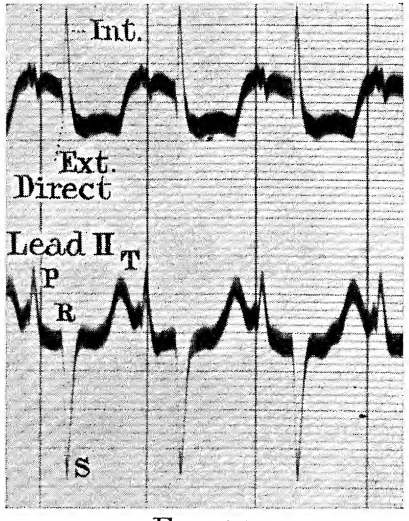


FIG. 14.

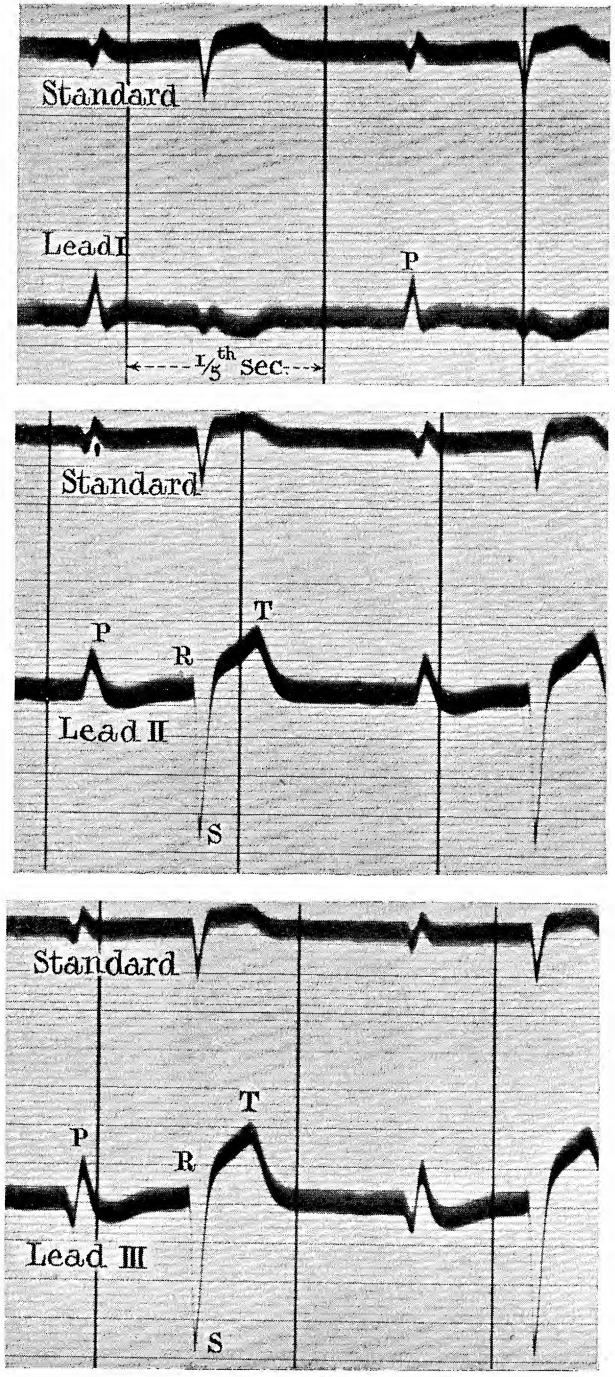


FIG. 15.

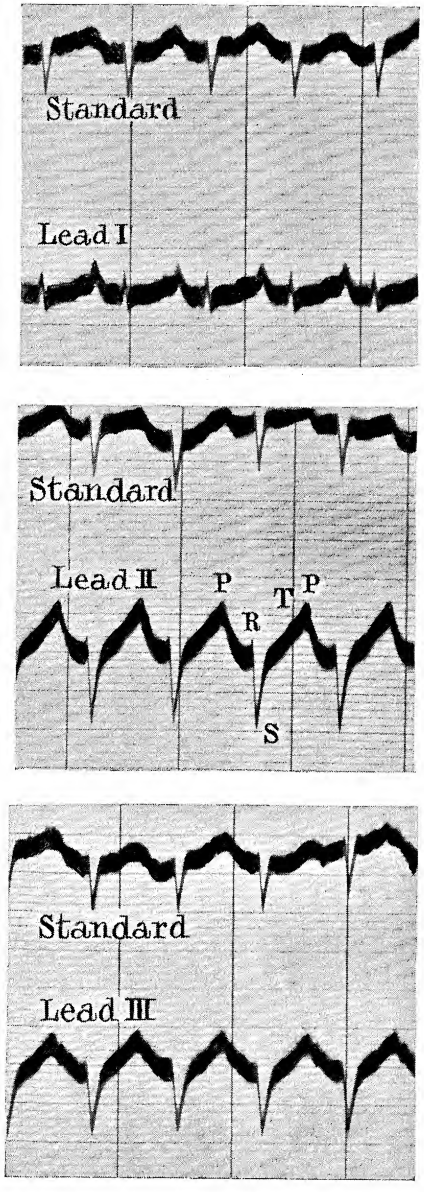


FIG. 16.

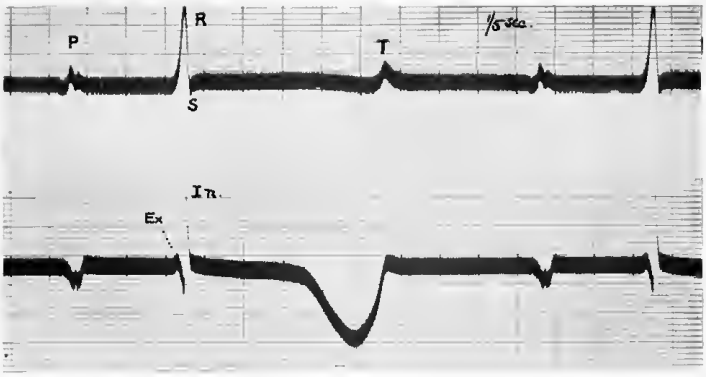


FIG. 7.

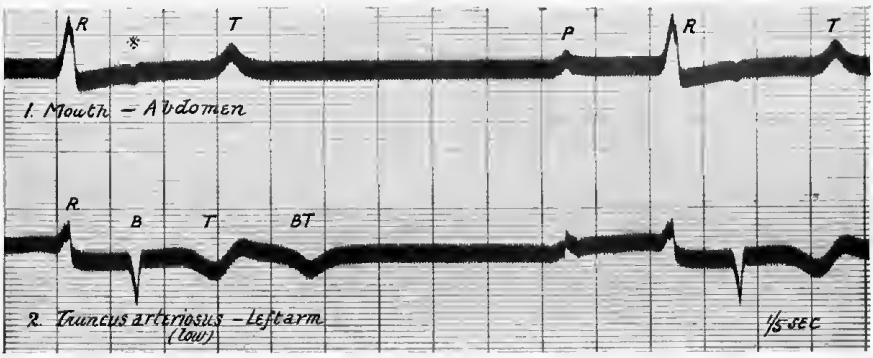


FIG. 8.

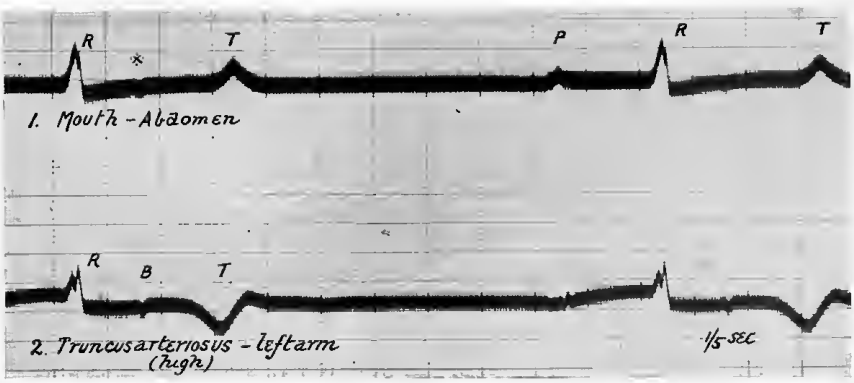


FIG. 9.

Fig. 7.—Toad *D*. Simultaneous curves (nat. size): (1) Lead *II*, from right side of pectoral girdle to mid-abdomen; (2) base of ventral surface of ventricle to body wall. The lower curve shows diphasic extrinsic deflections (*Ex.*) followed by a steep intrinsic deflection (*In.*). The reading of the intrinsic deflection is obtained by estimating the interval between the summit of *R* in the upper curve and the commencement of the intrinsic deflection in the lower curve, and subsequently relating it to the commencement of *R*. The vertical lines represent 0·2 second intervals.

Fig. 8.—Simultaneous curves (nat. size). (1) Standard lead from mouth to abdomen; this shows *R* and *T*, the ventricular summits, and a little downward notch marked *, which is due to the passage of the excitation wave through the *truncus arteriosus*. (2) Lead from *truncus arteriosus* to left arm region; this shows extrinsic deflections, which may be recognised as corresponding approximately to *R* and *T*, and intrinsic deflections *B* and *BT*. The *upstroke* of *B* corresponds to the arrival of the excitatory process under the *truncus* contact, *BT* to its subsidence at the same point. The *downstroke* at *B* is an extrinsic effect of the truncus itself.

Fig. 9.—Simultaneous curves from the same animal. (1) The top lead has been maintained. (2) The *truncus* contact has been moved off the musculature of the *truncus* to the commencement of the artery (for actual contact see fig. 4). This curve is consequently different from that of fig. 8. *R* and *T* and another small extrinsic deflection *B* remain, but the intrinsic deflections, *i.e.* the greater part of *B* and the whole of *BT*, have disappeared.

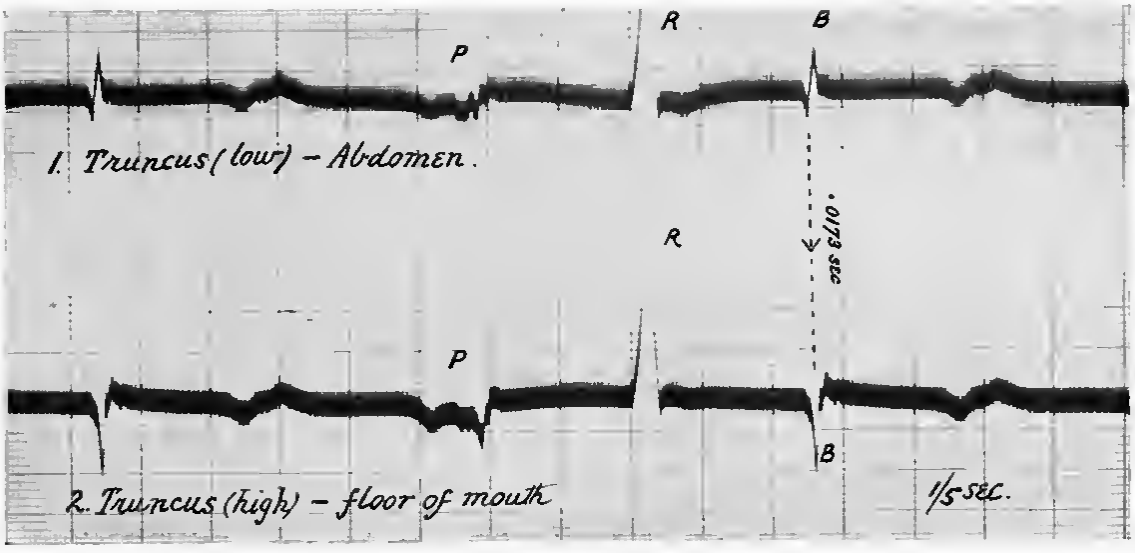


FIG. 10.

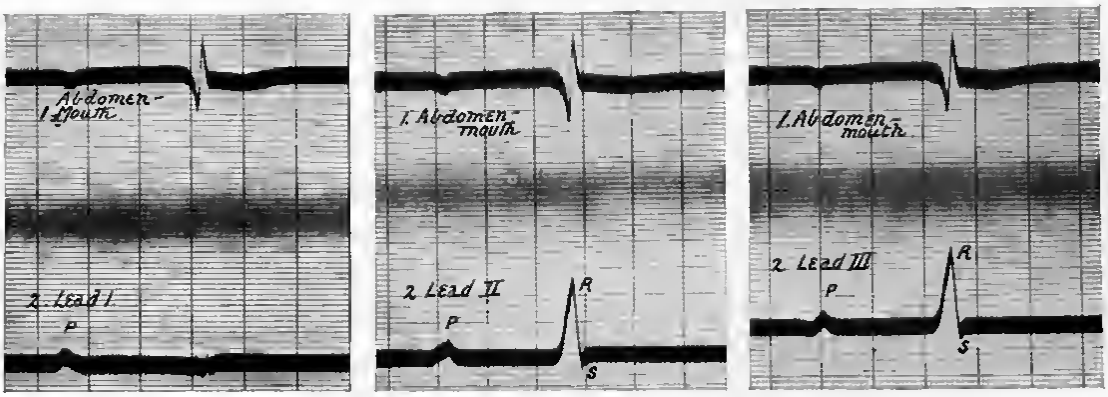


FIG. 11.

Fig. 10.—Simultaneous curves (nat. size) showing the method of estimating conduction rates. Two contacts are placed on the *truncus arteriosus*, above and below, and each is paired with a separate and indifferent contact on the body wall. The time interval between the intrinsic deflections (first sharp upstroke, *BB*) of the *truncus* is estimated. In this instance it amounted to 0·0173 second. The *truncus* contacts lay 2 mm. apart.

Fig. 11.—Three simultaneous curves ($\frac{2}{3}$ nat. size) from Toad *B*. In each photograph the upper curve is a standard curve from which to measure. The lower curves are taken from Leads *I*, *II*, and *III* (see fig. 3) respectively. The sensitivity of this string was such that 1 centimetre's excursion (10 scale divisions) corresponded to 1 millivolt. Time marker = 0·2 second. These three curves were utilised in constructing fig. 3 and Table V, Toad *B*.

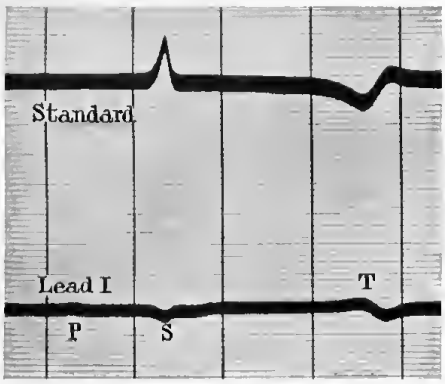


FIG. 4.

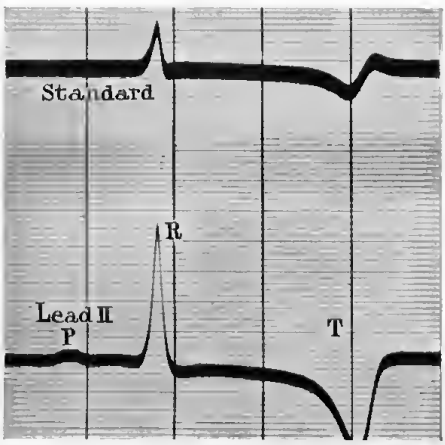


FIG. 5.

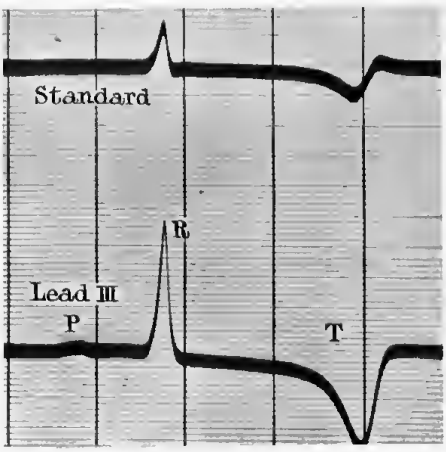


FIG. 6.

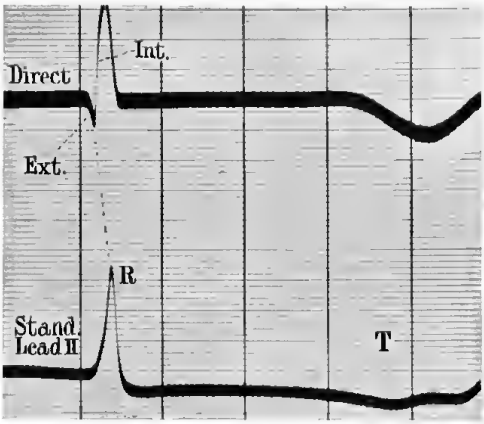


FIG. 7.

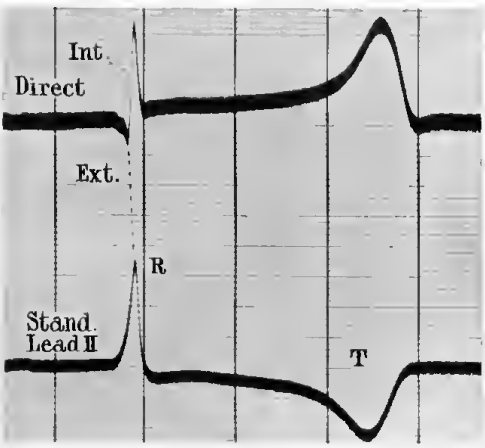


FIG. 8.

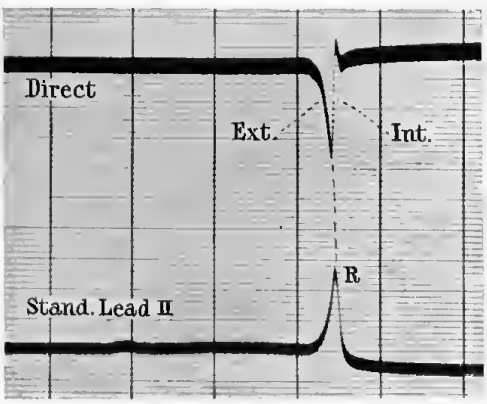


FIG. 9.

EXPLANATION OF PLATE 17.

Figs. 4, 5, and 6 (Tortoise *G*).—In each of these photographs the upper curve is the standard curve from the neck to a point outside the right and caudal aspect of the ventricle, the lower curves are from the leads *I*, *II*, and *III* of the triangle.

Ordinates of lower curves, 1 cm. = 1 millivolt; abscissæ = 0·2 second.

Figs. 7, 8, and 9 (Tortoise *G*).—Fig. 7: A direct lead from the lining of the ventricle near the *A–V* ring and an indifferent point on the body wall, taken simultaneously with Lead *II*. Fig. 8: Similar curves, the direct lead being from the ventricular surface near its centre. Fig. 9: Similar curves, the direct lead being from the lining of the heart near the ventricular apex.

In each direct lead an extrinsic deflection (*ext.*) and an intrinsic deflection (*int.*) is shown. The extrinsic deflection is very deep in fig. 9, but the intrinsic deflection rises from it sharply.

Ordinates of direct curves, 1 mm. = 1 millivolt; of lower curves, approximately, 1 cm. = 1 millivolt; abscissæ, 0·2 second.

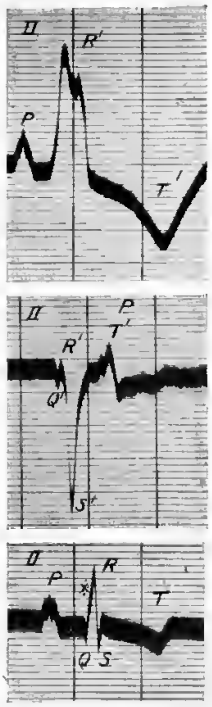


FIG. 12.

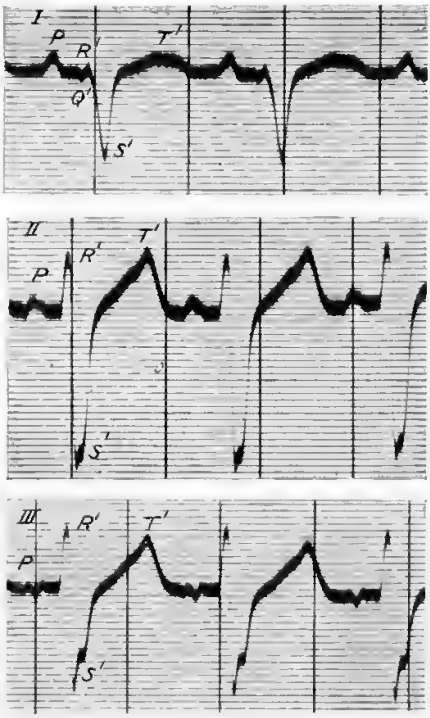


FIG. 13.

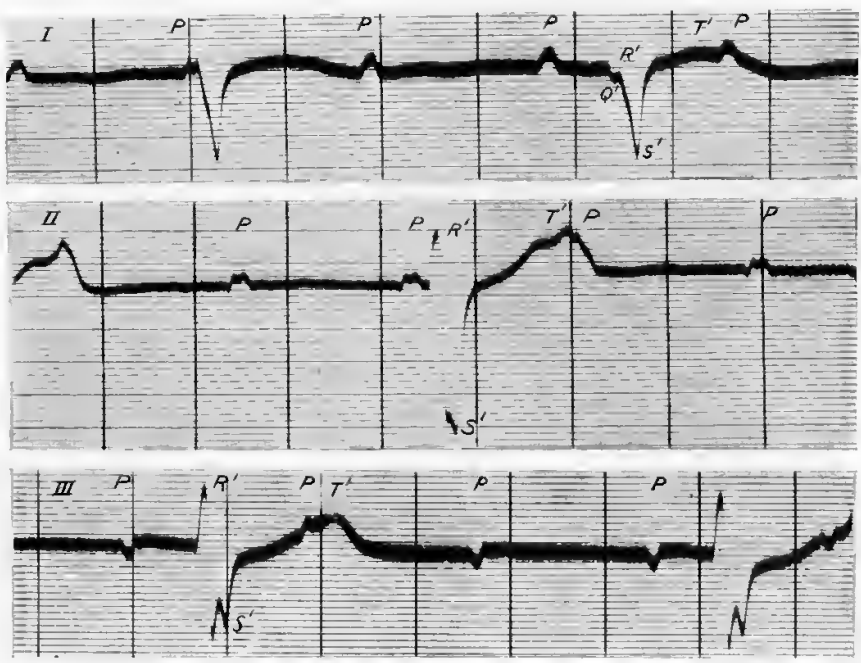


FIG. 14.

PLATE 18.

Fig. 12.—(Dog H.H.) Three electrocardiograms from Lead *II*. Top curve dextrogram, middle curve levogram, bottom curve bigram. While the dextrogram and levogram were recorded, complete heart-block was present. These three curves were used in the construction of fig. 2 (p. 251). Ordinates, approximately, 1 cm. = 1 millivolt; abscissæ = 0·2 sec.

Fig. 13.—Levograms from Leads *I*, *II*, and *III* (Dog I.O.), the result of transection of the right bundle division. Ordinates, approximately, 1 cm. = 1 millivolt; abscissæ = 0·2 sec.

Fig. 14.—The corresponding levograms, after clamping the *A–V* bundle and producing complete heart-block. The outlines of the ventricular curves in this and the preceding figure are essentially the same. Ordinates, approximately, 1 cm. = 1 millivolt; abscissæ = 0·2 sec.

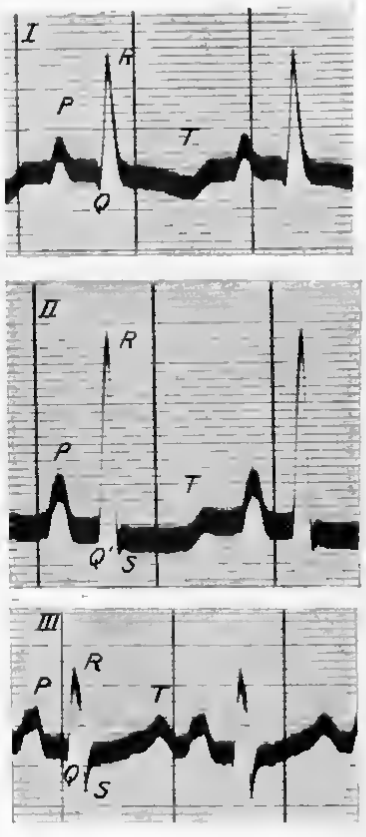


FIG. 15.

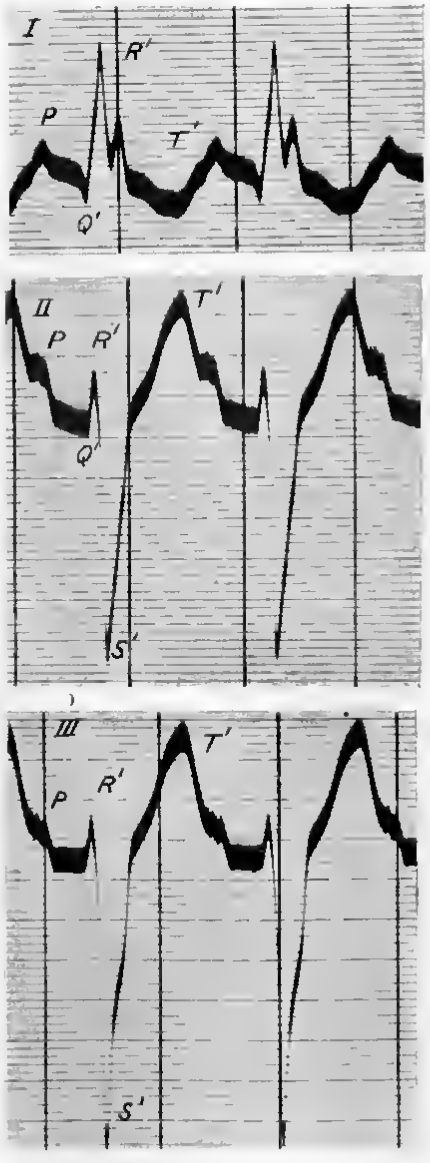


FIG. 16.

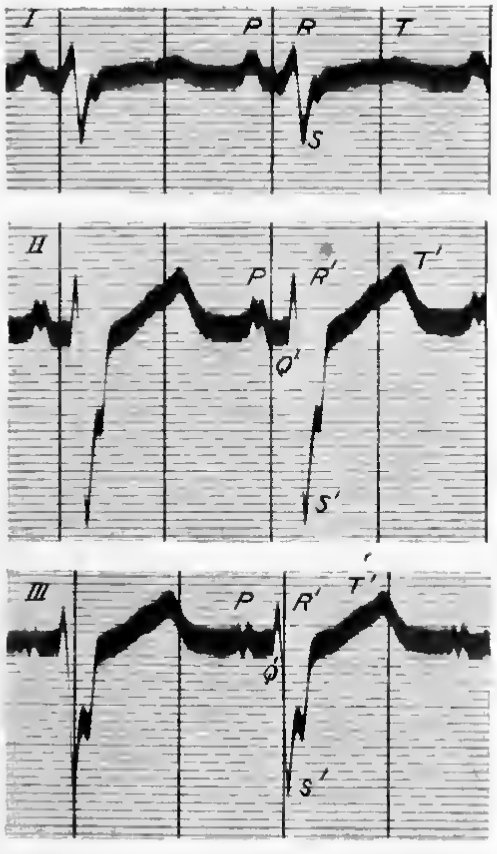


FIG. 17.

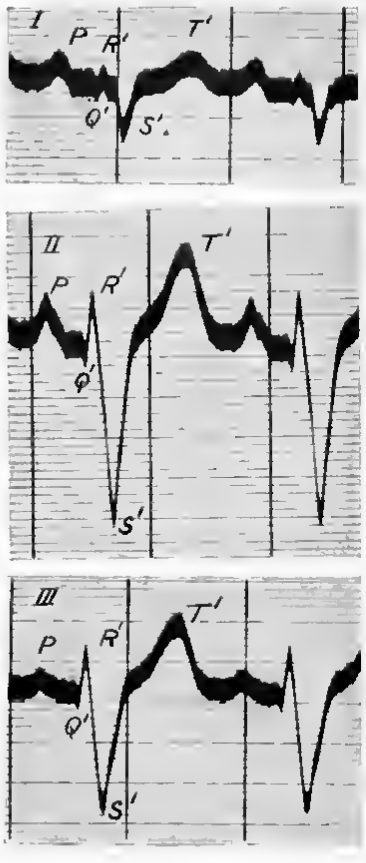


FIG. 18.

PLATE 19.

Fig. 15.—(Dog H.W.). Electrocardiograms from Leads *I*, *II* and *III*, chest wall open. Showing the curves of the natural heart beat. Ordinates, approximately, 1 cm. = 1 millivolt; abscissæ = 0·2 sec.

Fig. 16.—(Dog H.W.). Electrocardiograms from Leads *I*, *II* and *III*, chest wall open. Showing the form of the levogram, obtained by clamping the right bundle division in the same animal. The curves are of the discordant type. Ordinates, approximately, 1 cm. = 1 millivolt; abscissæ = 0·2 sec.

Fig. 17.—(Dog I.J.) Electrocardiograms from Leads *I*, *II* and *III*, chest wall open; levograms of concordant type produced by transection of right bundle division. Ordinates, approximately, 1 cm. = 1 millivolt; abscissæ = 0·2 sec.

Fig. 18.—(Dog H.P.) Electrocardiograms from Leads *I*, *II* and *III*, chest wall open; levograms of concordant type produced by clamping right bundle division. Ordinates, approximately, 2 cm. = 3 millivolts; abscissæ = 0·2 sec.

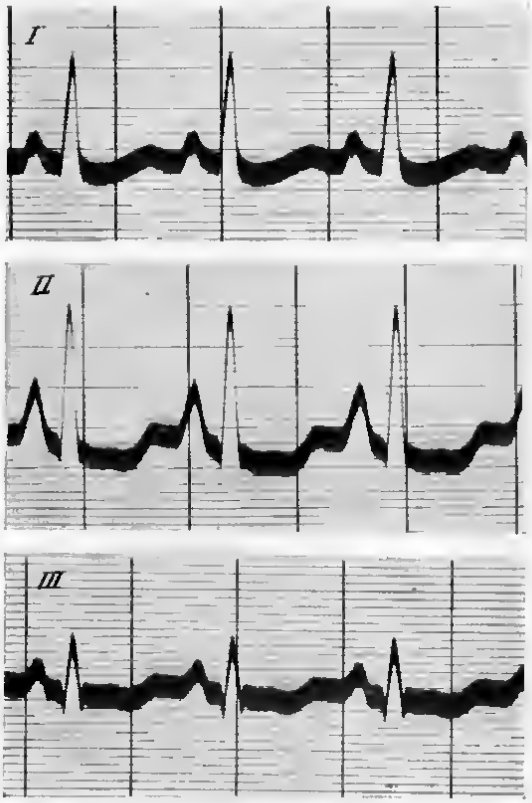


FIG. 19.

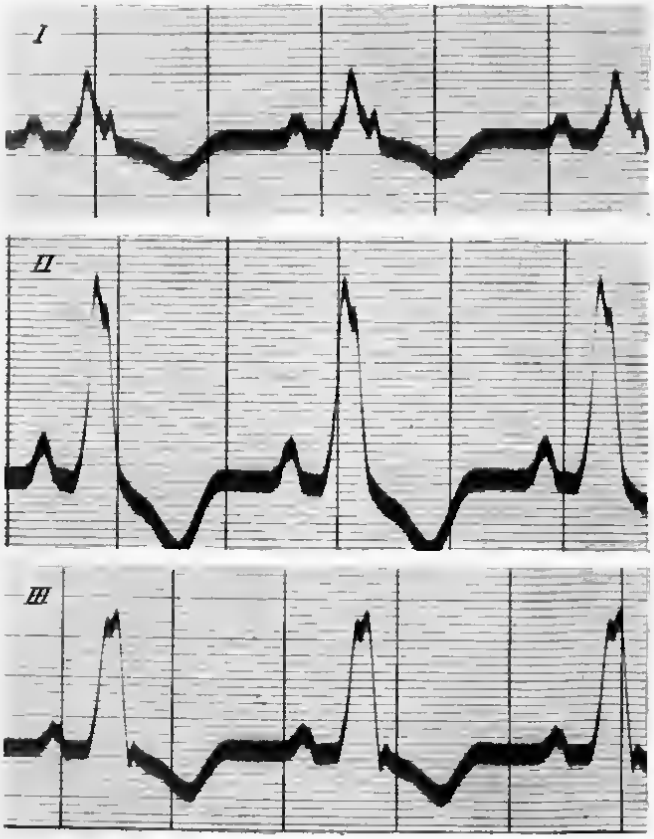


FIG. 20.

PLATE 20.

Fig. 19.—(Dog I.C.) Electrocardiograms from Leads *I*, *II* and *III*, chest wall open. Showing the curves of the natural heart beat. Ordinates, approximately, 1 cm. = 1 millivolt; abscissæ = 0·2 sec.

Fig. 20.—(Dog I.C.) Electrocardiograms from the same animal and from Leads *I*, *II* and *III*, showing the form of the dextrogram, produced by transection of the left bundle division. Chest wall open. Ordinates, approximately, 1 cm. = 1 millivolt; abscissæ = 0·2 sec.

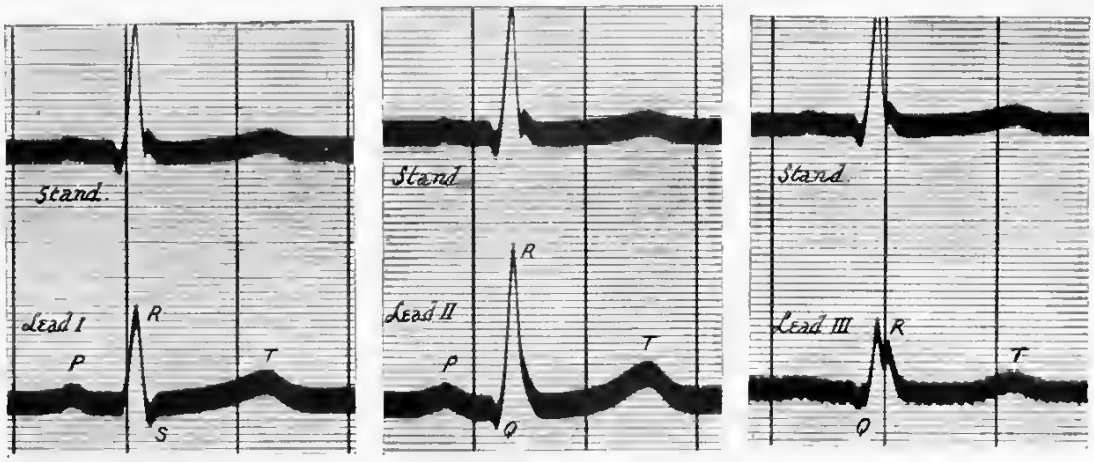


FIG. 10.

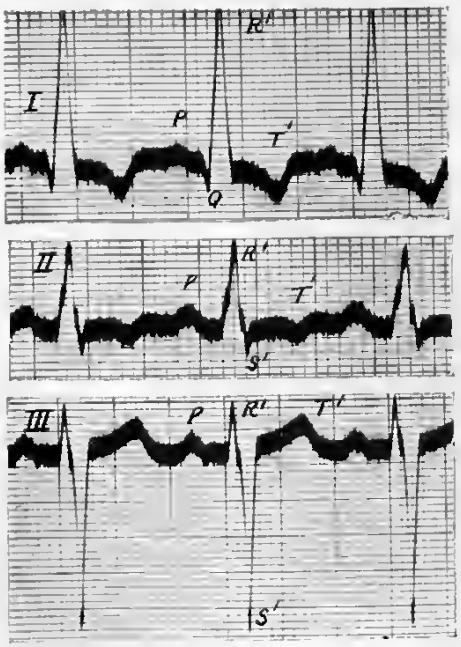


FIG. 11.

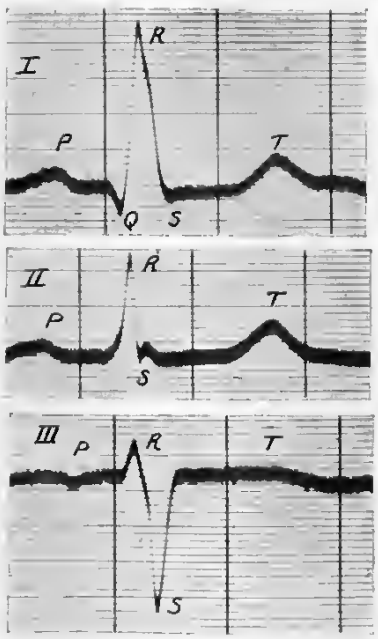


FIG. 12.

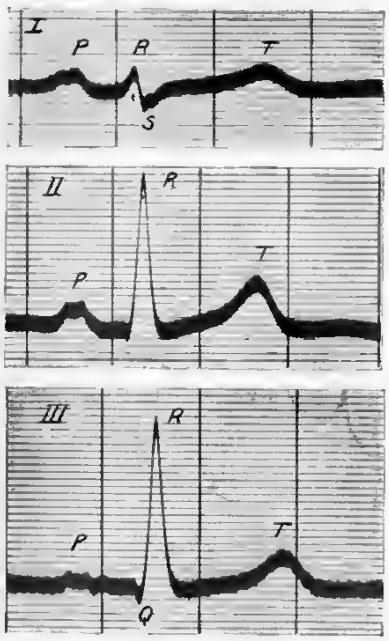


FIG. 13.

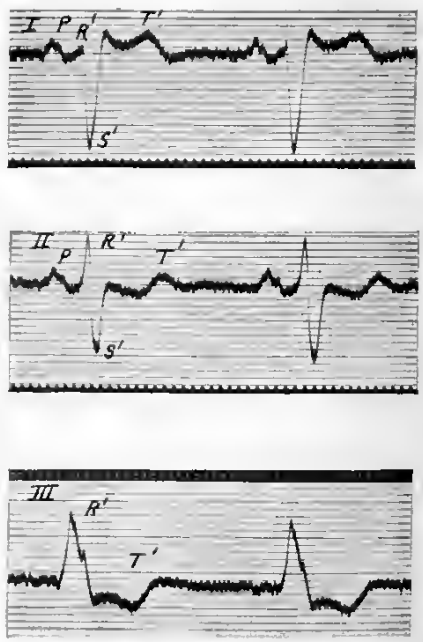


FIG. 14.

EXPLANATION OF PLATE 21.

Fig. 10.—Three photographs from a normal human subject. The upper curve in each is the standard lead, a fixed lead from the chest wall. The lower curves are from Leads *I*, *II*, and *III*. Ordinates, 1 cm. = 1 millivolt; abscissæ, 0·2 sec. These curves were used in constructing the chart of fig. 7.

Fig. 11.—Electrocardiograms from the three leads, indicating a right bundle branch lesion. Ordinates, 1 cm. = 1 millivolt; abscissæ, 0·2 and 0·4 sec. These curves were used in constructing the chart of fig. 1.

Fig. 12.—Three electrocardiograms from a patient suffering from aortic disease, and published for comparison with those of fig. 11. The resemblance is close, so far as the initial phases of the two sets of curves are concerned. Ordinates, 1 cm. = 1 millivolt; abscissæ, 0·2 sec. Used in constructing the chart of fig. 4.

Fig. 13.—Three electrocardiograms from a case of mitral stenosis. Ordinates and abscissæ as in fig. 12. Used in constructing the chart of fig. 6.

Fig. 14.—Three electrocardiograms from a patient in whom a defect in the left bundle branch is supposed to have existed. Ordinates, 1 cm. = 1 millivolt. Time marker in 0·03 sec. Used in constructing the chart of fig. 2.

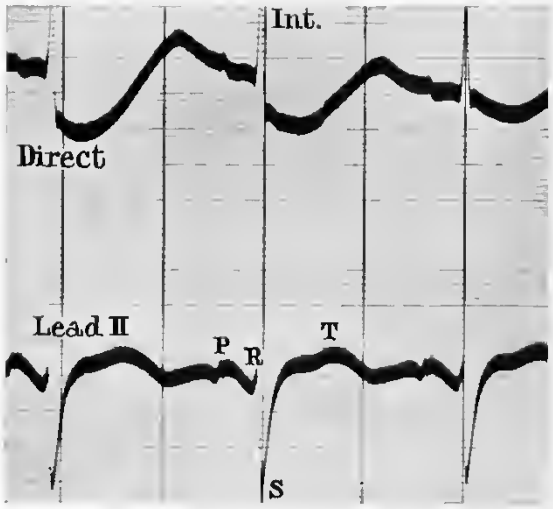


FIG. 13.

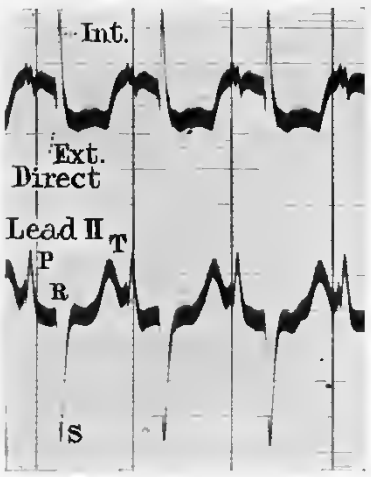


FIG. 14.

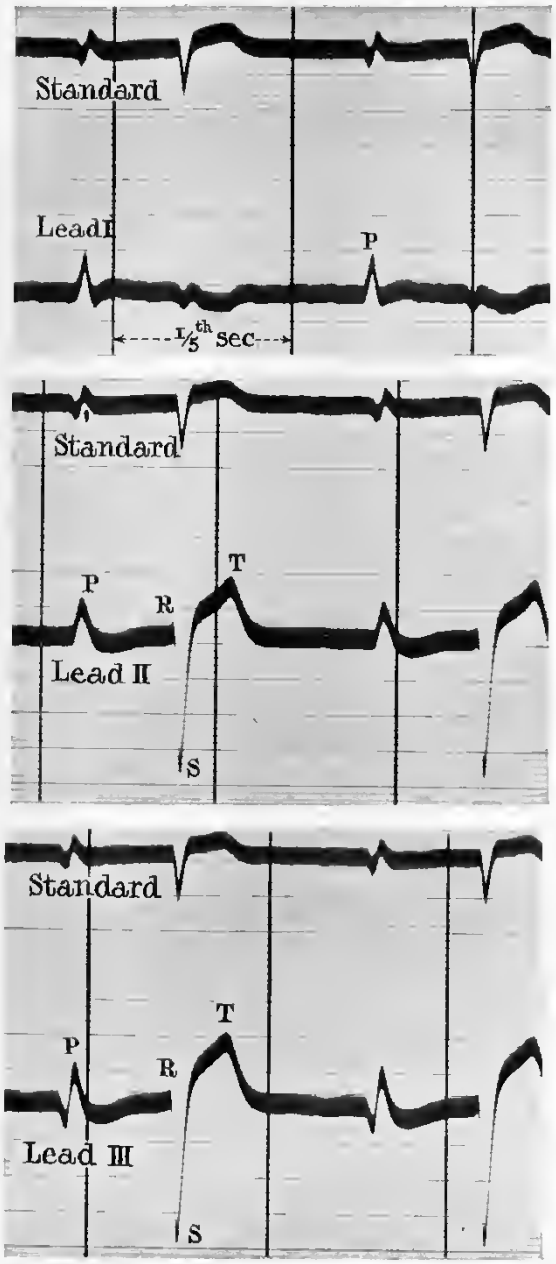


FIG. 15.

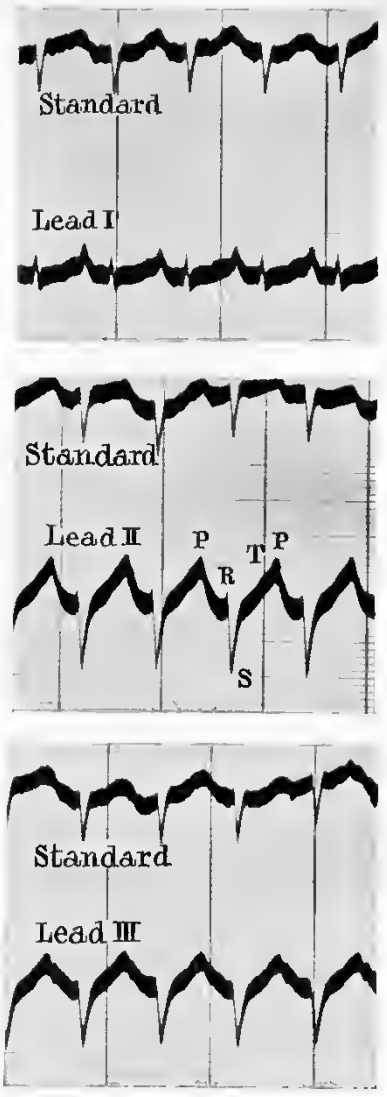


FIG. 16.

EXPLANATION OF PLATE 22.

Fig. 13.—(Fowl C.) Simultaneous electrocardiogram from Lead *II* and curve from direct ventricular lead. Illustrating the method of obtaining direct readings from the heart. The curve from the direct lead shows a minute extrinsic and a sharp and tall intrinsic deflection. Ordinates, for Lead *II*, approximately 1 cm. = 1 millivolt ; for the direct lead 1 mm. = 1 millivolt ; abscissæ = 0·2 sec.

Fig. 14.—Similar curves from a swan. In the direct lead, the extrinsic deflection is a distinct and sharp downward deflection and this is followed by the usual and prominent intrinsic spike. Ordinates and abscissæ as in fig. 13.

Fig. 15.—(Pigeon J.) Leads *I*, *II* and *III*, each taken simultaneously with a standard curve. The latter was from the top of the sternum to the lower part of the right chest wall. Ordinates of the three leads, approximately, 1 cm. = 1 millivolt ; abscissæ = 0·2 sec.

Fig. 16.—(Fowl I.) Similar photographs from Fowl I. Ordinates of the three leads, approximately, 3 cm. = 1 millivolt ; abscissæ = 0·2 sec.